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Tongue Muscle Spindle Afferents in Humans

by

© Catherine Fletcher

A THESIS

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled Tongue Muscle Spindle Afferents in Humans

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ABSTRACT

This study undertook to discover whether the muscle spindle fibers from the tongue of humans travel to the brain along the lingual nerve (a branch of the trigeminal nerve) or along the hypoglossal nerve. Considerable controversy has surrounded this question. A technique which utilized a characteristic response property of muscle spindles (a monosynaptic reflex followed by a silent period) was used. Electrical stimulation applied to the tongue of human subjects elicited the response. EMG recording techniques were used to record the response. The results of these experiments, while providing data pertaining to other areas of current experimental interest, were judged to be not clear enough to answer the question of interest here. A second technique was used. This technique made use of the characteristic monosynaptic reflex response of muscle spindles. It was elicited by mechanical stimulation of the tongue. The EMG response again was recorded. Following nerve block anesthesia of the lingual nerve the monosynaptic reflex response could no longer be elicited. It was therefore concluded that muscle spindle afferent fibers from the tongue of humans travel to the brain in the lingual nerve.

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INTRODUCTION

I am interested in the question of whether lingual muscle spindles are involved in the motor control of speech and if so in what way. However, this is a relatively untouched topic of investigation, and some basic anatomical and physiological questions must be answered before the more obviously linguistic questions can be posed. For example, a commonly-used technique for determining feedback function involves disruption of that feedback. In order to use this technique to determine the function of lingual muscle spindles in speech motor control it is necessary to determine how muscle spindle functioning can be disrupted. Using local anesthetic techniques there are in principle three ways of disrupting lingual muscle spindle feedback; (1) the nerve containing the muscle spindle efferents (the hypoglossal nerve) can be anesthetized, (2) the nerve containing the muscle spindle afferents can be anesthetized, or (3) anesthesia can be applied directly to the surface of the tongue. The question becomes, then, whether any or all of these techniques is practical.

Possibility (1), it will be argued, is a difficult and possibly unworkable procedure, involving selective anesthetic techniques. In order to evaluate whether or not possibility (2) is practical it is necessary to know by which pathway lingual muscle spindle afferents travel. There are two possibilities; they may be contained in the lingual nerve or they may be contained in the hypoglossal

nerve. This study is intended to determine which of these two possibilities actually holds. If in fact it is determined that lingual muscle spindle afferents travel in the hypoglossal nerve, then lingual muscle spindle afferent blocks will involve a procedure similar to the one that possibility (1) above involves. If, on the other hand, it is determined that lingual muscle spindle afferents travel in the lingual nerve, then lingual muscle spindle afferent blocks will be very easy to perform but unfortunately will involve anesthesia of the other afferents in the lingual nerve as well. It may, however, be possible to selectively anesthetize the lingual nerve so as to achieve anesthesia of only muscle spindle afferents (in other words, to use a procedure similar to the procedure mentioned when discussing possibilities (1) and (2) above).

Possibility (3) above will also be examined. In relation to this possibility the following questions will be considered: (a) is muscle spindle functioning disrupted?, and if the answer to this question is affirmative, then (b) is the disruption specific to muscle spindles?

While the purpose of this thesis has been expressed in terms of furthering the area of inquiry into the functioning of lingual muscle spindles in speech motor control the experiments outlined may be seen in other lights as well. Obviously experiments designed to determine by which pathway the lingual muscle spindle afferents travel can be seen to answer a purely anatomical question. From yet another

viewpoint, within the context of work that has been done on speech motor control in which lingual nerve blocks or surface anesthesia were used to achieve lingual tactile disruption, these two experiments can be seen to serve a janitorial function as well. That is, these experiments will serve to specify more fully what effects these two procedures have on oral sensation, thereby allowing us to be more confident in our interpretations of experiments which have used these techniques (and oblige us to re-interpret some experiments as well).

CHAPTER I

LITERATURE REVIEW

In this chapter I will first discuss in general terms the type of research done by linguists and speech scientists on the function of lingual feedback and the function of oral proprioceptive feedback in speech motor control. The purpose of this discussion is primarily to motivate the idea of doing basic physiology and anatomy as a linguist. The servo-assisted model of motor control will then be discussed, for the purposes of (1) emphasizing the importance of the role played by muscle spindles in the control of movement and (2) explicating the basic properties of muscle spindle functioning on which rest the experimental technique being used here. Next, some of the literature concerning the physiology and anatomy of various of the oral reflexes will be discussed, in two parts. In the first part some of the oral reflexes will be described and the mechanisms proposed to underlie them will be mentioned. This part will primarily be concerned with the results from experiments in which EMG techniques have been used. Therefore the EMG technique will also be discussed, including the EMG silent period technique (the technique being used in the present study). The second part will be concerned with the questions of the anatomical pathways of the oral reflexes. The final section of the chapter will be a review of the literature concerning the question of the

afferent pathway of muscle spindles from the tongue.

THE SPEECH CONTROL LITERATURE

Virtually all of the work which has been carried out on speech motor control has been done by linguists and speech scientists. Exteroceptive feedback and auditory feedback have been extensively studied, whereas proprioceptive feedback has received less attention. I will discuss the types of research that linguists and speech scientists have done on lingual exteroceptive and oral-facial proprioceptive feedback.

Tactile feedback from the tongue has been studied most extensively by "the Purdue group" speech scientists at Purdue University. The Purdue group's research strategy can be summarized in the following manner: elimination of tactile feedback by nerve block anesthesia of the nerve carrying the tactile afferents; acquisition of speech data from the subject both before and after anesthesia; description of the speech data primarily by qualitative means (close phonetic transcriptions, evaluations by panels of speech scientists and speech therapists); comparison of speech data obtained under the two conditions; and relating of specific anesthetic-related changes directly to a supposed function of tactile feedback. Results they have obtained have generally been described as consisting of sub-phonemic alterations of speech; slowing down of speech rate, distortions and substitutions, and changes in vocal

intensity, as examples. Interpretations of these and other results have varied. Initially it was felt that evidence had been provided that tactile feedback is an important feedback source which is utilized in speech motor control (Ladefoged 1967). In recent years members of the Purdue group have provided a different interpretation, stating that the fact that the speech changes found under various conditions of disrupted tactile feedback are sub-phonemic indicates that tactile feedback is only of slight importance in speech motor control. They propose an open-loop speech control model with closed-loop refinements of articulatory gestures (Scott and Ringel 1971).

There are numerous methodological and conceptual problems with the approach taken by the Purdue group, some of which are specific to this group and some of which are more generally associated with the majority of speech motor control research done by linguists. While these problems do not relate directly to this thesis, since this thesis does not concern tactile feedback, a discussion of these problems will serve to indicate the rationale for the present study.

There is a serious problem in the Purdue group studies concerning what the immediate effects of the anesthesia are. This general problem has at least two "origins"; one is the fact that nerve block anesthesia is not strictly local. There is always a certain amount of diffusion, and nearby nerves may be affected as well as the nerve which we want to anesthetize. Borden, Harris and Catena (1973) have shown,

for example, that even when extreme care is taken to avoid anesthetizing the mylohyoid nerve when anesthetizing the lingual nerve, it is quite unlikely that one will succeed in anesthetizing the lingual nerve alone. Another origin is the fact that much of the oral-facial neuroanatomy has not been determined; thus which sensory afferents and efferents are affected by anesthesia of many particular cranial nerves is not known. This problem is serious for several reasons; (1) without knowing which sensory afferents are anesthetized we do not know what to relate changes in the output of the system to in any specific terms, and (2) if efferent nerves are affected by the nerve block anesthesia, either by anesthetic diffusion or as a result of uncertainty as to the presence of efferents in the nerve to be anesthetized, then motor functions of muscles directly involved in speech production may be affected, and any changes in the output of the system may be wrongly attributed to the afferents presumed affected. As stated previously, the present study can be seen as an attempt to eliminate a part of one of these problems; namely to eliminate the problem of the controversy concerning the course of the lingual muscle spindle afferents. Stated in terms which relate more directly to the Purdue group studies, the present study will answer the question of whether or not the lingual muscle spindle afferents are anesthetized when the lingual nerve is anesthetized (lingual nerve anesthesia is a technique commonly used by the Purdue group).

A second problem that is present in the Purdue group studies might be termed "underestimation of the distant effects of anesthesia". In the Purdue group studies the characteristics of feedback-deprived speech are related directly to a supposed feedback function. No allowance is made for the possible effects due to such factors as the following: (1) non-specific stress induced in the subject as a result of the experimental situation, (2) adaptive control mechanisms (seen here to be "unconscious compensations"), and (3) conscious efforts to compensate for a perceived difficulty in articulating normally. Furthermore, no distinction is made between how the feedback information is used at each of the various neural levels. For example, it is known that trigeminal tactile afferents synapse on trigeminal motoneurones. Therefore disruption of trigeminal tactile afferents will "automatically" produce changes in the system output in the sense that it will alter the EMG activity in some trigeminal muscles, unless perhaps compensations are possible and are made for this feedback input change from higher structures. Presumably <u>that</u> type of disruption of the working of the speech motor control system is usefully distinguished from the type of disruption which may be caused by disrupting the normal functioning of the cerebellum and of the cerebral cortex, in speech motor control. It certainly is not obvious how these distinctions can be made when the output variable being examined is speech.

This same argument can be looked at from the opposite direction. Because theories of how feedback could be used in speech motor control have been so general, so non-commital with respect to the neural structures and levels involved, expectations concerning what sorts of changes in speech output should occur under conditions of sensory deprivation have not been derivable from them. Consequently the technique of collecting speech data under conditions of feedback disruption have largely been unmotivated theoretically. Questions arise as to whether the correct parameters in the speech signal were selected for analysis. (Note that just exactly this problem was what brought the Purdue group to using such techniques as close phonetic transcriptions and panels of judges to analyze the data.) Interpretations seem to be arbitrary because there seem to be no ways of deciding upon the significance of observed "effects". (Recall here that the interpretations of the Purdue group results have varied over the years in spite of relatively consistent findings.) But in addition to the difficulties created by the lack of a theory there is also the fact that experimenters have perhaps naively assumed that any and all functions of feedback in speech motor control will be reflected in some way in the speech output data obtained under sensory deprivation and that the nature of the speech changes will be logically linkable to those functions.

Another recognizable group of scientists who are

interested in the relationship between oral sensation and perception, and speech motor control is "the Bosma group". The Bosma group consists of experimenters of various backgrounds who have met on several occasions (Bosma 1967, 1970). They have, for the most part, attempted to determine whether there is a clear relationship between oral stereognostic perceptual skill and speaking ability. The assumption underlying these studies has been that oral stereognostic perception must require the proper functioning of all of the oral sensory modalities and furthermore, the proper functioning of the integration of the information from these modalities. A further assumption is that these functions (the modalities themselves and their integration) must be the same functions necessary for normal speech production. The Bosma group studies suffer, then, as a result of any criticisms which may be made of these assumptions. In addition, they suffer from the criticisms made above of studies which use speech as the dependent variable because for the most part speech is the dependent variable examined in these studies. Not surprisingly, results from these studies have been controversial.

It is useful to summarize the similarities and differences between the research carried out by dental physiologists and that carried out by linguists. While to a large extent these two groups of scientists are interested in the functioning of the same structures, orientations and methodologies are for the most part distinct. Dental

physiologists have been oriented toward examining the reflex mechanisms which are likely to be involved in motor control of chewing. Linguists, on the other hand, have been oriented toward testing non-physiologically-based models of speech motor control, lumping any possible reflex mechanisms which might be operating together with any other mechanisms which might be operating in the feedback-deprived subject while he is speaking. Or, stated in slightly different terms, the dental physiologists have been mapping the sensory-motor pathways linking various of the oral-facial structures (i.e., doing anatomical work) and determining the nature of these connections, with a view toward discerning their functions in motor control of chewing (i.e., doing physiological work). Linguists have been attempting to relate their work to abstract functional models of speech motor control largely without regard for the anatomical structures and physiological mechanisms involved.

The most reasonable conclusion to draw at this point seems to be to adopt a new approach. The new approach requires (1) constructing a theory of how feedback could be used by the speech motor control system which is physiologically interpretable and (2) using physiological measures of the output of the system (in other words stepping back one or two steps in the linguistic communication process sequence, from the acoustic waveform to the activity of the neuromuscular structures which produced the waveform). Some researchers (other than those

in the Purdue group, that is) have begun to examine what may be presumed to be the mid-brain effects of anesthesia, by examining EMG as the output variable instead of speech (Borden 1972; Borden et al 1973; Abbs 1973; Abbs, Folkins and Sivarajan 1975). This tack seems to be much more straight-forward and interpretable.

In contrast to their great interest in the role of oral exteroceptive feedback in speech control the Purdue group has consistently been uninterested in the role of muscle spindle feedback. This lack of interest is reflected in the following naive statement made by Scott in 1970 (p. 27).

> To the writer's knowledge, the controversy concerning the existence of muscle spindles in the human tongue has not been completely settled.

(The existence of muscle spindles in the human tongue was demonstrated by Cooper in 1953 and confirmed by Walker and Rajogopal in 1959.)

What work has been done on the role of proprioception in speech motor control has primarily concerned laryngeal proprioceptors. There is only one study I know of in which the primary purpose of the study was to determine the role of proprioception in the control of some oral-facial muscle during speech. That is the Abbs (1973) study in which selective anesthesia of the mandibular nerve was the technique used to determine the muscle spindle role in the control of jaw movements during speech. (The selective anesthesia technique will be discussed later.) Abbs

concluded on the basis of that study that he had found support for the hypothesis of spindle involvement in speech motor control. There has been no such work done on the role of muscle spindles in the tongue. It is worthwhile to consider possible reasons why this is so.

While Abbs was able to selectively anesthetize the mandibular nerve and thereby to isolate the gamma efferents from the large diameter alpha efferents and large diameter afferents, use of an equivalent technique on the hypoglossal nerve may not be practical. First, bilateral anesthesia of the hypoglossal nerve is a dangerous procedure which could cause swallowing of the tongue. The question can thus be raised concerning whether unilateral nerve blocks result in sufficient disruption of feedback to satisfy the purposes of the speech scientist. Second, unilateral hypoglossal nerve anesthesia can only be effected at an anatomical location inferior to the tongue, on the floor of the mouth. In this position the right and left hypoglossal nerves are very close to each other and therefore unilateral blocking of hypoglossal nerve is difficult. These facts may constitute reasons why a procedure similar to the Abbs procedure has not been used in evaluating muscle spindle functioning in the tongue. I have found no discussions of this in the literature, however.

One could still consider the possibility of anesthetizing the muscle spindle afferents. In fact, one could argue, anesthetizing the muscle spindle afferents will

eliminate all muscle spindle functioning whereas anesthetizing the muscle spindle efferents will only reduce their functioning by eliminating gamma bias; therefore afferent fiber anesthesia is to be preferred anyway. But, as has already been pointed out, the question of the lingual muscle spindle afferent pathway has not yet been settled for humans.

It is clear that some experimenters have felt that they had anesthetized the muscle spindle afferents when they anesthetized the lingual nerve. Ladefoged (1967: 165), commenting on an experiment done by Ringel and Steer (1963) in which lingual blocks were performed, said

> they arranged for their subjects to be at least partially deprived of kinesthetic as well as tactile feedback from the tongue by anesthetizing the fifth cranial nerve, which carries the sensory impulses from these muscles.

Other experimenters have realized the uncertainty involved in assuming that lingual muscle spindle afferents travel in the lingual nerve (Putnam 1973; Scott 1970).

The question of the spindle afferent pathway to the central nervous system is important to linguists for two reasons (1) if the lingual nerve contains spindle afferents then the experiments in which lingual nerve blocks have been performed under the assumption that it contained no proprioceptive afferents, must be re-interpreted, and (2) knowing the pathway of the spindle afferents, we may be in a better position to devise ways of singling out

proprioceptive afferent information from other types of afferent information and better assess its role in speech motor control.

It may be pointed out that the question of the pathway of the lingual muscle spindle afferents is by no means the only controversial anatomical question of interest to linguists. For example, it is not clear whether or not there are motor fibers present in the infraorbital nerve. The Purdue group have used the technique of infraorbital nerve block anesthesia in order to determine the role of tactile feedback from the lips in speech motor control (Putnam 1973; Ringel 1962; Ringel and Steer 1963; Scott 1970; Scott and Ringel 1971) under the assumption that the nerve did not contain motor fibers. Abbs et al (1975) have recently completed a study in which tests of "magnitude and rate of upper lip displacement (for speech and non-speech tasks), the magnitude and rate of upper lip depression force, and diadochokinetic rate" were performed before and after bilateral infraorbital nerve block anesthesia in order to determine whether there were motor fibers present in the infraorbital nerve. Although they found some inter-subject differences which they attributed to inter-subject anatomical differences they concluded that they had found sufficient evidence of the presence of motor fibers in the infraorbital nerve. These results vitiate the earlier results from experiments in which infraorbital nerve blocks were employed.

MOTOR CONTROL

Introduction

Motor control in general has long been an area of great interest to physiologists. Several different bases (or sets of structural and functional principles) for motor control have been found. The set of principles of motor control operative in a particular species has been found to be related to its position in the phylogenetic scale. thus, for example, typical amphibian muscle spindles are different from typical mammalian muscle spindles and some of the concepts developed in relation to describing amphibian motor control may not be extended to describing mammalian motor control (Matthews 1964). Even within the same species more than one basis for motor control can be found; thus, for example, muscle spindle afferents from mammalian hind-limbs do not project to the cerebral cortex, whereas those from the fore-limbs do (Oscarsson and Rosen 1963). Most work on motor control has been done on non-humans, which raises questions concerning the extent to which the results may be extended to accounting for motor control in humans. In addition, most motor control research has been done on the spinally-innervated musculature. With the exception of the thoracic musculature the speech musculature is cranially innervated. There are several differences between the cranial nerves and spinal nerves, which raises further questions concerning the generality of these results in terms of accounting for speech motor control. Still

further, while some work has been done on the physiology of the structures used in chewing (mostly done by dental physiologists), some of which has been done on humans but most of which has been done on experimental animals, there are obvious differences between speech and chewing functions. The significance of the difference between speaking and chewing, for the purposes of those interested in investigating speech and language functions, is twofold. First, the types of cortical and cerebellar activities involved in speaking are not the same as those involved in chewing.[1] Second, even when asking questions pertaining to the lower structures involved in chewing (presumably much the same structures as are used in speaking), those interested in chewing functions may not ask those questions which are linguistically interesting.

However, it would be foolish to dismiss physiological models of motor control (that is, models constructed by physiologists) on the grounds of their irrelevance for speech motor control. On the contrary it is a heuristic of biological research to use models of the functioning of biological systems which are derived from study of phylogenetically lower species as hypothesized simplified models of the functioning of analogous systems in higher species. At the same time we must grant the caveats mentioned above and be cautious in extending physiological models of motor control to speech motor control.

In discussing physiological models of motor control we

first want to distinguish between central and peripheral control of movement. Study of central control of movement primarily involves study of the roles of the cerebral cortex and of the cerebellum in motor control. One of the differences between speech and other motor activities mentioned earlier is a difference in the involvement of the cerebellum in the control of these two classes of movements. Bowman and Combs (1969b) have provided evidence that the cerebellum is not involved in speech motor control to nearly the extent that it is in the control of other types of movement. Direct cortico-motoneuronal pathways exist from the cerebral cortex to the speech motoneurones in humans (Bowman 1971). An analogous situation appears to operate in the control of finger muscles in humans and other primates as well (Bowman 1971; 43). In addition, they (Bowman and Combs) claim (1969b) that lingual muscle spindle afferents, unlike limb spindle afferents, do not project to the cerebellum. Rather, they project to the ventrobasal thalamic complex (Bowman and Combs 1969a), and from there directly to the cerebral cortex (Bowman and Combs 1969b). Bowman accounts for these and other such facts in terms of a theory of encephalization; of motor control of highly-learned, skilled, and phylogenetically high activities by the cerebral cortex instead of by both the cerebral cortex and the cerebellum. These facts and interpretations are intriguing in themselves, but I want to distinguish these and other aspects of central motor control of speech from

the topic of this thesis, which comes under the rubric of peripheral motor control. Peripheral motor control involves the motor and sensory mechanisms which operate at a sub-cortical and sub-cerebellar level. Obviously, as is the case with most useful distinctions, the central-peripheral distinction is somewhat arbitrary.[2]

Muscle Spindles

In models of the peripheral control of movement the muscle spindle plays a major role. Perhaps as a consequence, most motor control research has centered around determining the capabilities of the muscle spindle feedback system. As an example, the excellent review of peripheral control of movement by Stein (1974) is essentially a review of research of muscle spindles, although mention is also made of structures such as Golgi tendon organs. What follows is basically a summary of the Stein review and the earlier review by P. B. C. Matthews (1964).

Although not all of the details of the morphology of muscle spindles appear to have been agreed upon, a fairly detailed picture arises out of the literature. Only the broad outlines will be presented here.

The muscle spindle consists of from two to twelve intrafusal muscle fibers. They normally attach at their ends to muscle fibers; thus they are arranged in parallel with the extrafusal muscle fibers. There are basically two types of intrafusal muscle fibers, although intermediate

types have been noted (Barker 1962). Both types have a large number of nuclei in their central portion: they are distinguished morphologically primarily in terms of how the nuclei are arranged. One type is the nuclear bag fibers, which are characterized as possessing a central bulge which contains the nuclei. The second type, nuclear chain fibers, have their nuclei arranged in a central row or chain. Both types are well striated at their ends and poorly striated in the central nuclear region (the nuclear bag fibers are particularly lacking in central striations). Nuclear bag fibers normally attach to extrafusal muscle fibers whereas nuclear chain fibers normally attach to nuclear bag fibers.

The two types of afferents innervating muscle spindles were described as early as 1898 by Ruffini. They were called primary and secondary endings. Primary endings are located in the central region of both nuclear bag and nuclear chain fibers. They have very large nerve fiber diameters (group IA); thus they conduct very rapidly. Secondary endings are located on the ends of the intrafusal fibers, with terminations on the nuclear chain fibers being more widely-branched than on the nuclear bag fibers. They have slightly smaller nerve fiber diameters (group II) than do the primary endings and thus do not conduct as rapidly.

Primary and secondary afferents are clearly differentiated on physiological as well as on anatomical grounds. Both types of muscle spindle afferents are stimulated to fire when the muscle spindle is being

stretched. Both primary and secondary endings signal the length of the intrafusal fiber but only the primary endings respond to the dynamic phase of a stimulus. That is, primary endings fire in response to the velocity of intrafusal fiber lengthening as well as to the absolute length of the intrafusal fiber. Secondary endings, then, are tonic receptors and primary endings are both tonic and phasic receptors. The relatively non-contractile nature of the central portion of the intrafusal fibers is thought to be significant in initiating and shaping the primary ending response (i.e., in producing the dynamic component of the response over and above the static component of its response).

The two now generally-accepted types of efferent innervation to the spindle were first conclusively differentiated by Matthews in 1962 (Matthews 1964, 1970). They are called dynamic and static fusimotor fibers. Both types are within the gamma range of nerve fiber diameters. The anatomical distinction between the dynamic and static fusimotor fibers is a topic of controversy, but the functional distinction is generally accepted. The difference between dynamic and static fusimotor fibers can be observed only by recording the discharges of muscle spindle afferent fibers while the muscle spindle is undergoing a length change and individual fusimotor fibers are being stimulated. Stimulation of static fusimotor fibers increases the tonic response of both the primary and

secondary endings while having little effect on, or decreasing, the primary afferent phasic response (Stein 1974; Matthews 1964). Stimulation of dynamic fusimotor fibers under these same conditions increases the velocity or phasic response of the primary afferents. At the time that the Matthews (1964) review was written there had been much discussion in the literature of the possibility that there was an alpha nerve supply to the muscle spindles in addition to the two gamma nerve supplies. Matthews considers the histological and physiological evidence in favor of such a view to be suggestive but not conclusive.

Muscle spindle afferents synapse with a variety of functionally-grouped alpha motoneurones. Muscle spindle group II afferent firing produces a flexor reflex; excitation of ipsilateral flexor muscles and inhibition of ipsilateral extensor muscles. The response is the same regardless of whether the group II afferents originate in a flexor or in an extensor muscle. The functional significance of this flexor reflex has not been discerned. A number of different reflexes are simultaneously mediated by group IA afferents. The stretch reflex is a monosynaptic excitatory reflex. It involves the group IA afferents and the alpha motoneurones of the same muscle. Thus the firing of group IA afferents from a particular muscle will monosynaptically excite the alpha motoneurones in the same muscle to fire. The group IA afferents also synapse monosynaptically, by collaterals, with the alpha

motoneurones innervating muscles which are synergistic (or agonistic) to the muscle of afferent origin. The nature of this connection is not excitatory (directly causing the alpha motoneurones to fire) as in the case of the stretch reflex, but is facilitatory (lowering the threshold of the alpha motoneurones) (Ruch et al 1965). The group IA afferents also inhibit the alpha motoneurones of muscles which are antagonistic to the muscle of afferent origin, via a disynaptic pathway. All of the reflexes take place at a single joint. These latter two types of reflexes are not always restricted to involving agonists and antagonists of the muscle of afferent origin (Matthews 1964).

In addition to their action on motoneurones, muscle spindle afferents may pre-synaptically inhibit activity in other afferent nerves (Nakamura 1971) and also may be inhibited themselves by the action of other afferent nerves (Matthews 1964).

Muscle spindles have been found in all of the spinally-innervated voluntary muscles in which they have been sought. There has been considerable controversy over whether they also exist in all of the human cranial voluntary muscles. There is dispute, for example, over whether they exist in all of the human jaw-closing muscles (Voss 1956; Smith and Marcarian 1967). The number of muscle spindles found in any particular muscle seems to be less related to the size of the muscle than to the extent to which it is subject to fine control. Thus, for example, in the cat's lateral

gastrocnemius muscle there are fewer muscle spindles than there are in its soleus muscle (35 versus 55), although the latter is a far smaller muscle (Matthews 1964). muscle spindles were found in the human intrinsic lingual muscles by Cooper in 1953, after decades of debate on the issue. Her finding was confirmed by Walker and Rajagopal (1959). Cooper (1953) studied the distribution of muscle spindles throughout the various lingual intrinsic muscles. It was found that there are few muscle spindles in the tongue tip region and that they are particularly dense in the transverse muscles away from the mid-line and in the superior longitudinal muscle near the mid-line. Muscle spindles are not present in either the extrinsic or intrinsic lingual muscles of non-primates (Boyd 1937; Carleton 1938; Weddell, Harpman, Lambley and Young 1940; Cooper 1953; Blom 1960).

Muscle Spindles and Motor Control

Currently the most widely-accepted theory of muscle spindle functioning is the theory of servo-assisted motor control. This theory proposes that the output originating from higher centers is co-distributed between the alpha and fusimotor fibers (Granit 1966; Stein 1974). The amount of stimulation distributed to the alpha motoneurones can be seen to be a first approximation to the amount of stimulation required tc produce the amount of muscular tension necessary in order to satisfy the motor command.

Then any discrepancies between the amount of stimulation provided and the amount required, such as might be brought about by muscular fatigue or by an unexpected external load, are handled by the muscle spindle system. If extrafusal contraction brought about by alpha stimulation procedes faster than "intended" by the higher centers then muscle spindle action will slow the contraction; if it procedes slower than "intended" then muscle spindle action will speed up the extrafusal contraction. Before it can be made obvious how this is said to operate in specific terms it is necessary to discuss how the various components of the muscle spindle morphology are thought to function.

First we must ask how the fusimotor system participates in motor control. If the gammas are not stimulated to an appropriate extent during extrafusal shortening the muscle spindles will become slack and cease to fire. Servo assistance to motor control is not operative under these circumstances. It is thought that gamma efferent stimulation functions in part to eliminate the cessation of feedback information. The muscle spindles can be kept "tight" by this gamma stimulation and thus extremely sensitive to any lengthenings of the extrafusal fibers. Thus the fusimotor system can control the muscle spindles' sensitivity to extrafusal fiber stretch. On the same basis the fusimotor system can control the rate of discharge of the muscle spindles in response to a stretch; the less fusimotor activity, the lower the frequency of discharge.

A second question we may ask is: what is the significance of the fact that muscle spindles are both phasic and tonic receptors? In servo terms, in order to damp oscillations in the output of the system about a desired mean an element must be built in which will predict future positions. This element will compensate for unavoidable time delays in the system that can result in oscillations. In the motor control system the delay is brought about by the muscle spindle feedback taking a certain amount of time before it is effective and by muscle sluggishness. The phasic response component of the primary ending response is thought to provide the information that is required in order to eliminate oscillations, or to damp the stretch reflex arc.

A third question is: what is the functional significance of the dual efferent innervation of the muscle spindle? Note that the fusimotor system can control the amount of damping of the stretch reflex. In servo terms this results in versatility of the system in responding efficiently to external loads of various sizes. In other words, stability is added to the system by its ability to increase or decrease the velocity response of the primary endings, depending upon the mass of the load to be moved.

In summary, the servo-assisted model of motor control depicts the muscle spindle as playing the role of providing the moment-to-moment adjustments of motor output required in the motor control system in order to compensate for changes

in the internal state of the muscle and/or changes in the external load on the muscle.

Because of the inherent capabilities of spinally-innervated muscle spindles in shaping the final response of muscles to movement demands, we would expect muscle spindles to be of much value in the motor control of finely-controlled voluntary movements such as are involved in speech motor control. Therefore linguists should be interested in the question of whether lingual (and other) muscle spindles participate in shaping the response of speech muscles.

ORAL REFLEXES

There is a growing body of recent dental physiological literature which can be seen to be an attempt to specify the oral-facial sensory-motor connections. The basic aim of these studies is to specify the mechanisms involved in normal mastication, and thus enable us to better diagnose and treat malfunctions and abnormalities. EMG techniques are commonly used in these studies. In this section some of the basic oral reflexes will be discussed. First, however, some general comments concerning EMG studies will be made.

EMG Studies

EMG studies involve stimulation of sensory receptors and observation of the effect of such stimulation on motor activity output, generally measured by EMG recordings of

muscle activity. Various sorts of sensory receptors have been stimulated, including periodontal receptors, pain receptors and muscle spindles. If there exists a neural connection between the receptor stimulated and the muscle being recorded from, this connection is reflected in a change in the EMG activity in the muscle; an increase if the connection is excitatory or facilitatory, a decrease if the connection is inhibitory or disfacilitatory. Muscle spindle stimulation causes a complex set of EMG responses in connected muscles. In the EMG response from the muscle of afferent origin a period of increased EMG activity (a stretch reflex) is followed by a silent period (when there is virtually no EMG activity). (Note that the term 'silent period' is a technical term. It is not to be confused with decreases in EMG activity not associated with a stretch reflex. When referring to the latter sort of decrease in EMG activity or to a period of decrease in EMG activity for which the cause is unknown I will use the term "period of EMG inactivity".) Agonistic muscles are facilitated (i.e., their threshold is lowered, but they do not necessarily fire) (Ruch et al 1965). Antagonistic muscles show only a period of EMG inactivity (no stretch reflex).

The factors involved in producing the period of EMG inactivity in antagonistic muscles are different from those involved in producing the silent period in the muscle of afferent origin. The period of EMG inactivity in antagonistic muscles is the result of inhibition carried by

the di-synaptic pathway connecting antagonistic muscles (referred to above). The silent period in the muscle of afferent origin is thought to be caused by two peripheral and one or two central factors (Granit 1955). The peripheral factors are (1) active inhibition carried by pathways longer than the pathway carrying the excitation which produces the stretch reflex, and (2) the cessation of excitation or facilitation. The active inhibition is thought to be mediated by the Golgi tendon organs (Granit 1955). The loss of excitation or facilitation corresponds to the cessation of muscle spindle afferent firing brought about by extrafusal muscle fiber contraction (referred to in the above discussion of muscle spindles). (Angel et al 1965). Huftschmidt and Spuler (1962) suggest that Golgi tendon organs may be involved in the production of the masseteric silent period following jaw tap and Bessette et al (1974) provide evidence that periodontal receptors provide a major source of inhibition in producing this period of EMG inactivity, and that disfacilitation or active inhibition from muscle receptors is insufficient to produce it. Bratzlavasky (1972a, c) provided good evidence that the silent period in the masseter muscle following electrical stimulation of the same muscle could be explained on the sole basis of the pause in the masseteric muscle spindle afferent discharge and that the reflex is restricted to the ipsilateral side. A central factor which may contribute to the production of silent periods is the autogenic inhibitory

action of recurrent collaterals of alpha motoneurones. While found to be a factor in spinal silent periods (Granit 1955; Renshaw 1940), recurrent collaterals have not been found in the trigeminal system (Lorente de No 1933) nor have they have been found for hypoglossal motoneurones (Lorente de No 1933; Porter 1965), although there is some evidence for Renshaw-type cell activity for jaw elevator motoneurones (Kidokoro, Kubota, Shuto and Sumino 1968b; Munro and Griffin 1970). A second central factor, influence from higher centers on the excitability of motoneurones, while capable of shortening or abolishing the silent period in peripheral muscles, does not affect the masseteric silent period (Bessette, Mohl and Bishop 1974).

By the use of the "silent period technique" it can be determined whether muscle spindles are present in any particular muscle (Bratzlavasky 1973). Stimulation of muscle spindles in the muscle leads to the production of a silent period in the EMG of that muscle. Therefore the presence of a silent period under these conditions is evidence of the presence of muscle spindles in that muscle.[3]

Similarly, by the use of the silent period technique it can be determined whether there is a reflex connection between the muscle spindle afferents of one muscle and the alpha motoneurones of another muscle, and the nature of the reflex (whether excitatory or disfacilitatory). The technique is not as simple as determining whether there is a drop in the EMG of one muscle correlated in time with

stimulation of another muscle, however. As mentioned above, receptors other than muscle spindles may give rise to EMG inhibition. Pain receptors are one such receptor. In distinguishing muscle spindle silent periods from inhibition brought about by receptors such as pain receptors several different criteria have been used. Some of these criteria are; the latency of the onset and duration of the EMG inhibition, the characteristics of the stimulus adequate to produce the inhibition, the relation of other experimental conditions to the nature of the response, and characteristics of the EMG response other than characteristics associated with the inhibition.

Many EMG techniques can be applied utilizing either human subjects or experimental animals. However, in some situations experimental animals must be utilized owing to the nature of the stimulation being used. And in other situations, for example those requiring a constant amount of muscular tension, it is more convenient to use human subjects. The results from EMG studies utilizing human subjects and those from EMG studies utilizing experimental animals are largely comparable. There are some differences, though. Therefore for ease of comparison I will discuss some of the results from animal experiments separately from the results from human experiments.

Sherrington (1917) discussed the mechanisms he felt to be responsible for the cyclicity of jaw movements: they were (1) the jaw-closing reflex and (2) the jaw-opening reflex.

In addition there is the jaw-jerk reflex. The jaw-closing reflex can be initiated by mechanical stimulation of the dorsum of the tongue or by the presence of fluid in the mouth. The jaw-opening reflex is initiated by mechanical or electrical stimulation of the gums or teeth. It is followed immediately by "rebound" jaw-closing. Sherrington's results are based largely on work done on decerebrate cats but partly also on humans (actually he used himself as his subject). More recent investigations have, for the most part, implicitly assumed that this "reflex model" of the cyclicity of jaw movements is valid, although there have been some attempts to account for cyclic jaw movements in terms of a "pace-maker model" (Magoun, Ranson and Fisher 1933; Sumi 1970; Dellow and Lund 1971; Denavit-Saubie and Corvisier 1972; Lund 1976; Thexton 1973b, c, 1974b, 1976).

The jaw-jerk reflex, a jaw-closing reflex, is a classical stretch reflex. Its existence has been confirmed many times (McIntyre and Robinson 1959; Goodwill 1968; Bessette, Bishop and Mohl 1971, Bessette *et al* 1974; Goldberg 1971; Munro and Griffin 1971; Hannam 1972; Fujii and Mitani 1973), its monosynaptic nature is well-established (Hugelin and Bonvallet 1956; McIntyre and Robinson 1959) and its pathway has been determined (Cajal 1909; Weinberg 1928; Corbin 1940; Corbin and Harrison 1940; Szentagothai 1948). The reflex is similar in humans and experimental animals. The jaw-jerk is evoked by sharply tapping the chin. The tap is thought to produce jaw

displacement, resulting in stretching of the jaw-closer muscles, which in turn unloads the muscle spindles in the closer muscles and brings about an autogenic stretch reflex (excitation of the jaw-closer muscles and disfacilitation of the jaw-opener muscles). The rebound closure described by Sherrington is thought to be brought about by the activation of a jaw-jerk reflex (Jerge 1964). The stretch reflex response has been measured in the masseter and temporalis muscles but no observations have been made on the medial pterygoid. The typical response in temporalis has been reported to be different from that in masseter (Munro and Griffin 1971). I will report only the results concerning masseter. Values reported for the latency in man of the excitatory response in the masseter muscle have varied between 3.5 msec and 12 msec.[4] The variation is partly the result of variation in the event used as the trigger for the measurement of the EMG, partly the result of variation in the methods used for analyzing and presenting data and partly the result of normal variation in the event itself. The latency and duration of the silent period following the stretch reflex varies a great deal from one experimental situation to another depending largely on the voluntary effort by the subject (whether the subject voluntarily opens his mouth immediately following tooth contact) (Hannam et al 1969). Munro and Griffin (1971) have reported a significant negative correlation between the latency and duration of the silent period. Bessette et al (1971, 1974) have given the

values of 20-31 msec (mean=24 msec) as the normal range of the values for the duration of the silent period.

The magnitude of the excitatory response is heightened if the subject maintains background EMG activity in the jaw-closer muscles (Fujii and Mitani 1973) in conformity with the fact that a stretch reflex is heightened when the gamma motoneurones innervating the muscle in which the excitation is found, are already being activated centrally. The duration of the silent period is unaffected by biting force (Bessette et al 1973).

The silent period in the jaw-closers and the inhibition in the jaw-openers can only be observed when background EMG activity is present in these muscles. Matthews (1975) has reported the latency and duration of the inhibitory response in the digastric muscle as 12 msec and 10 msec, respectively.

A response pattern similar to the one produced by jaw tap (at the same latency and with the same shape) can be produced by tapping various parts of the head and face. This response is called the jar reflex (Sherrington 1898). It is thought that the vibration set up by the tap causes the jaw-closer muscle spindles to unload, producing an autogenic stretch reflex (Hannam et al 1970). Muscle spindles have been shown to be extremely sensitive to vibratory stimulation (Brown, Engberg and Matthews 1967; Matthews and Stein 1969). A tonic vibration reflex can easily be evoked in man by the application of a vibratory

stimulus (100-200 Hz) to the chin (Bratzlavasky 197). Its origin is likely muscle spindles.

There is considerable agreement among a large number of recent experimenters that stimulation of sensory nerves serving the intraoral mucosa of cats produces a short-latency (9 msec latency peak) hyperpolarization response followed by a second hyperpolarization response (40 msec latency peak) in the masseter muscle (Goldberg and Nakamura 1968; Kidokoro, Kubota, Shuto and Sumino 1968a; Gura, Limanskii and Pityavskii 1969; Iwata, Sakai and Deguchi 1971; Goldberg 1972b; Takata and Kawamura 1973). There is general agreement that the first wave of hyperpolarization is an inhibitory post-synaptic potential (IPSP) and that it involves at least a dy-synaptic pathway. The second phase of hyperpolarization clearly involves a polysynaptic pathway involving a different set of interneurones (Kidokoro et al 1968b; Goldberg and Nakamura 1968; Sumino 1971) but it is not clear what mechanism produces the hyperpolarization (cf. Goldberg and Nakamura 1968; Kidokoro et al 1968a; Gura et al 1971; Iwata et al 1971; Sumino 1976). The amplitude of the second hyperpolarization continues to increase with increases in stimulus intensity (to the inferior alveolar or lingual nerve) to up to 7 or 8 times threshold (Kidokoro et al 1968a) whereas the amplitude of the first hyperpolarization increases with increases in stimulus intensity only up to about 2.5 times threshold (Kidokoro et al 1968a; Goldberg 1972b).

It has also been shown that the masseteric monosynaptic reflex is depressed in two phases, corresponding to the two phases of hyperpolarization of masseteric motoneurones, following stimulation of intra-oral sensory nerves (Sumino 1971; Goldberg 1972b).

A period of depolarization of masseteric motoneurones occurring between the two phases of hyperpolarization at a latency of approximately 18-20 msec or longer in response to electrical stimulation of intra-oral sensory nerves of cats has also been reported (Goldberg and Nakamura 1968; Kidokoro et al 1968a; Sessle and Greenwood 1976). Sometimes this depolarization results in spike production. The depolarization has been found to be due not solely to the reversal of the early hyperpolarization (Sumino 1971). It is thought (Goldberg 1972b) that the second hyperpolarization normally masks this depolarization.

Sherrington (1906) showed that a jaw-opening reflex would become a jaw-closing reflex if inhibitory mechanisms were blocked by strychnine or tetanus toxin. Considerable experimental interest has been shown in recent years in this jaw-closing response. Depending on the mode of excitation used and perhaps the central state of the experimental animal as well, this jaw-closing response is more difficult or less difficult to obtain than the simultaneously activated first phase of hyperpolarization (as is illustrated in some of the experimental results reported below).

Kidokoro et al (1968a) and Sumino (1971), using anesthetized cats, have obtained results similar to those of Sherrington by stimulating the inferior dental and inferior alveolar nerves respectively and then intravenously injecting strychnine. The early hyperpolarization was changed to a depolarization at the same latency.

A short latency jaw-closing response can be elicited without the use of inhibition-blocking drugs. Takata and Kawamura (1970) reported depolarization of masseteric motoneurones preceding any observable hyperpolarization at a latency of 2.5 msec, following electrical stimulation of the maxillary nerve in cats, and their photographs show the same response following stimulation of the inferior alveolar nerve (1971).

Sumino (1971) has also found in cats, that a conditioning stimulus applied to the inferior alveolar nerve in some cases (4 out of 10) caused a slight facilitation of the masseteric monosynaptic reflex immediately before the first period of depression. Sumino (1971) has concluded on the basis of transection experiments, that the excitatory interneurones involved in this reflex are located in the nucleus oralis, the nucleus interpolaris or the adjacent reticular formation and project at most dy-synaptically to the motoneurones of the ipsilateral jaw-closing muscles. Goldberg (1976) has observed short-latency depolarization responses in the masseter muscle in response to inferior dental nerve stimulation in anesthetized cats. In one case

spike initiation occurred at a latency of 2 msec. Goldberg (1972a) has reported elicitation of a short-latency excitatory response in the masseter muscle of cats and monkeys following electrical stimulation of the lingual nerve (in the cats) or of the gingiva (in the monkeys). This response preceded any response in the digastric muscle. The latency of the response was 5 msec in cats and 9 msec in monkeys. The response could be obtained, in the cats, when the jaw was held in an open position or when the jaw was held elevated, with the teeth in occlusion. It could only be obtained, in the monkeys, when the jaw-closing muscles were in isometric contraction. Under other conditions (e.g., when the jaw was at rest) only a longer-latency excitatory response was seen in the masseter muscle. Its latency was 16-18 msec. Goldberg (1972b) found that the first phase of depression of the masseteric reflex produced by a single electrical shock delivered to either the contralateral or ipsilateral lingual nerve of anesthetized cats was replaced by facilitation after midline section of the brain stem between the trigeminal motor nuclei.

Transient jaw closing in response to light mechanical stimulation of a large (2 cm^2) area of the hard palate, to the sudden release of a slow-onset light mechanical stimulation applied to a small (0.03 cm^2) area of the hard palate (Thexton 1968, 1973a) or to carefully controlled electrical stimulation of the mucosa (Thexton 1974a) or exposed infraorbital nerve of decerebrate cats has been

reported recently. Mechanical stimulation of sudden onset and greater force, and light mechanical stimulation of sudden onset and release applied to a small area of the palate produced jaw-opening and prolonged application of force to a large area of the palate (even of light force) produced prolonged jaw-opening. Digastric activity was sometimes present along with jaw-closure when the stimulation consisted of rapid onset and rapid release of the mechanical stimulus[5] but was never seen when the closure was induced by the sudden release of a slow-onset stimulus. In the case of the electrical stimulation of the exposed infraorbital nerve, temporalis excitation can be achieved by stimulating only the lowest threshold fibers (10-12 microns diameter), while simultaneous temporalis and digastric excitation can be achieved by increasing stimulus strength, without an immediate reduction of the temporalis response (1974a). The latency of the EMG activity in temporalis in the jaw-closing reflex was 7-9 msec when the stimulus used was mechanical (1973a) and 5-6 msec when the stimulus used was direct electrical stimulation of the (infraorbital) nerve (1974a). When the digastric activity was present during a jaw-closing reflex it occurred at the same latency as the temporalis response (1973a, 1974a) and was followed by a second (stronger) burst of temporalis activity with a latency of between 12 and 20 msec (1974a). Matthews (1975) has found that the jaw-closing reflex is most easily elicited when a brief, light mechanical force is applied to

a small (2 mm^2) area of the incisive papilla. He found the latency of the response in masseter is 5 msec, with no response in the digastric muscle. The response was potentiated if a stretch reflex in masseter was simultaneously evoked. Electrical stimulation of the same site failed to elicit the reflex (Matthews 1975).

Harrison and Corbin (1942) reported finding a jaw-closing reflex in response to tapping the zygoma or other bony prominence of decerebrate cats. They considered this response to be a jar reflex. The possibility that the jaw-closing reflex reported by Thexton (1973a) could have been not the result of stimulating palatal receptors but the result of eliciting a jar reflex was examined by Thexton by anesthetizing the palate by local injections of lignocaine. The jaw-closing reflex disappeared (Thexton 1973a).

A single electrical pulse applied to the maxillary mucosa or to the maxillary nerve of decerebrate cats (Thexton 1968), or to the infraorbital nerve of decerebrate cats (Thexton 1974a)), painful electrical or mechanical stimulation of intraoral sites (Thexton 1973a) or certain types of innocuous mechanical stimulation of the palatal mucosa of cats (Thexton 1973a) will produce jaw-opening. (Sessle and Greenwood (1976) have reported finding excitation of digastric motoneurones and inhibition of masseter motoneurones in response to tapping a tooth of anesthetized or decerebrate cats or monkeys. The inhibition in masseter motoneurones was in one early phase at low

stimulus levels and was in two phases, one early and one late, at high stimulus levels). It has been reported that jaw-closing will result if a single pulse is followed by a train of electrical pulses each sub-threshold for achieving jaw-opening (Thexton 1968). Mechanical stimulation of the palate immediately after a jaw-opening reflex will also cause jaw-closure (Schoen and Koeppen 1931). Reflex jaw opening initiated by moderate strength electrical stimulation can also be converted into jaw closing if it is initiated during an opening movement (Thexton 1974a) (the latency to temporalis activity is 5-6 msec), but is not convertible if the opening mevement is initiated by a strong stimulus.

Sessle et al (1973) have reported preliminary results from experiments in which they found an excitatory effect of periodontal receptors on jaw-closer motoneurones in cats. Sessle and Greenwood (1976) have found that tooth tap in anesthetized or mid-collicular decerebrate cats can excite alpha and gamma motoneurones of jaw elevator muscle. Twenty-five out of thirty-nine masseter alpha motoneurones tested showed the excitatory response (mean latency=6.1 msec, s.d.=1.40 msec) and 8 out of 10 masseter gamma motoneurones tested showed the excitatory response (mean latency=6.7 msec, s.d.=1.68 msec). Recordings were made from single neurons in the motor nucleus of the trigeminal nerve. Sessle and Greenwood favor the view that a monosynaptic pathway is suggested, especially given the

latency to masseter alpha excitation. Responses were depressed or abolished following local anesthesia around the stimulated tooth. Local anesthetic could also reversibly abolish the period of EMG inactivity following tooth tap. Matthews (1975) has reported a very powerful jaw-closing reflex which can sometimes be elicited in decerebrate cats by maintaining firm pressure on an upper canine or molar tooth while the mouth is being opened. No response is obtained if the mouth is not opened. Matthews conjectures that the excitatory effect of the stretch reflex in the closer muscles lowers the threshold of excitability of the jaw-closer motoneurones sufficiently that the excitation produced by the stimulus on the tooth is sufficient to fire the jaw-closer motoneurones.

Funakoshi and Amano (1974) have obtained two different periods of increased jaw muscle activity in response to lightly pressing or tapping the maxillary incisor of anesthetized albino rats. The first response had a latency of 7 msec and the second response had a latency of 10-60 msec. Responses were largest in the ipsilateral masseter muscle. Both responses were eliminated following transection of the ipsilateral maxillary nerve. The second response disappeared after section of the brain stem between the motor nucleus and the main sensory nucleus of the trigeminal nerve. The first response disappeared following transection between the mesencephalic nucleus of the trigeminal nerve and the motor nucleus of the trigeminal

nerve. The first response had a lower threshold, as determined be eliciting the reflexes by electrical stimulation of the mesencephalic nucleus of the trigeminal nerve. By recording latencies of evoked potentials along the reflex arc it was determined that the pathway mediating this reflex is polysynaptic.

Several studies have reported on the presence of a crossed reflex period of EMG inactivity in the masseter muscle of man produced by mechanically (Schaerer, Stallard and Zander 1967; Brenman et al 1968; Ahlgren 1969; Griffin and Munro 1969; Munro and Griffin 1970; Hannam et al 1969, 1970; Goldberg 1971; Sessle and Schmitt 1972) or electrically (Goldberg 1971) stimulating a tooth or by electrically (Hoffman and Tonnies 1948; Bratzlavasky 1972a, c, 1973; Yemm 1972a, b) or mechanically (Matthews and Yemm 1970; Bratzlavasky 1972c; Yu et al 1973) stimulating other oral sites. Its latency is approximately 13 msec and its duration is approximately 15-20 msec.[6] Some studies have reported that this period of EMG inactivity is followed by a second period of EMG inactivity (Bratzlavasky 1972a, c, 1973; Yemm 1972a, b; Yu et al 1973). The latency of this response is approximately 40-60 msec.[7]

Between the first and second periods of EMG inactivity a period of increased EMG activity has been reported. Bratzlavasky (1972c), Matthews (1975) and Sumino (1972) have all pointed out the similarity in the time courses of the two periods of EMG inactivity in the masseteric motoneurones

found in experiments utilizing mechanical or electrical stimulation of oral sites in humans, and the two phases of hyperpolarization of masseteric motoneurones following electrical stimulation of intraoral sensory nerves of non-humans.

Some have suggested that the first period of EMG inactivity following mechanical tooth stimulation is an inhibitory period and that stimulation of periodontal ligament mechanoreceptors is responsible for it (Brenman, Black and Coslet 1968; Ahlgren 1969; Griffin and Munro 1969, 1970). However, the fact that the response is still present when mechanically stimulating the tooth after anesthesia of the tooth (Hannam, Matthews and Yemm 1970; Goldberg 1971), the fact that the same response is obtained by eliciting a jaw-jerk or a jar reflex (Hannam et al 1973) and the fact that the response is present in subjects wearing full dentures (Matthews and Yemm 1970) have cast doubt on this interpretation. These latter observations suggest that perhaps the period of EMG inactivity found was due at least in part to the eliciting of a jar reflex and thus is constituted of, at least in part, a silent period. This interpretation is supported by the fact that preceding the period of EMG inactivity in masseter after tooth tapping or tooth contact there is a period of biphasic synchronous activity in the masseter motoneurones (Hannam et al 1970; Goldberg 1971). Hannam et al (1970) claim that this period of activity corresponds in latency to the latency of the

jaw-jerk reflex. That the first period of EMG inactivity is only in part constituted of a silent period is suggested by the fact that the response in masseter after anesthesia of the tooth is of shorter duration than before anesthesia, but it is still present (Hannam et al 1970; Goldberg 1971). (Goldberg reports the duration before anesthesia as 15.0-29.1 msec (mean=20.1, s.d.=6.0 msec) and the duration after anesthesia as 8-20 msec (mean=12.2, s.d.=4.1 msec).)

Evidence has been provided in several recent investigations that the peak of EMG activity preceding the first period of EMG inactivity following mechanical stimulation of a tooth is not simply a jaw-jerk reflex, as suggested by Hannam et al (1970), but rather that the first period of EMG inactivity is preceded by a different excitatory response in masseter (in addition to the jaw-jerk reflex, in the case of mechanical stimulation). Goldberg (1971) has found that delivering an electrical pulse to the gingiva over the root of the central incisor of human subjects produces an excitatory response in the ipsilateral masseter muscle which he has termed the periodontal-masseteric reflex (Goldberg 1971). Sessle and Schmitt (1972) have found a similar excitatory response in man following tooth tap. Goldberg has measured its latency as being 6.5 to 8.8 msec (mean=7.5 msec, s.d.=0.9), approximately 2.5 msec shorter than his own measurements of the latency of the jaw-jerk. (The values he obtained for the latency of the jaw-jerk are 8-12 msec (mean=9.0 msec,

s.d.=0.9).) Contrast this with the results of Hannam et al (1970) that the excitatory response in the masseter muscle to mechanical stimulation of a tooth has the same latency as does the jaw-jerk reflex. Goldberg found the excitation to be followed by a period of EMG inactivity with a duration of 15.0-29.1 msec (mean=20.1 msec, s.d.=6.0). No response could be seen unless background activity was present. When anesthesia was applied to the region of the tooth being stimulated electrical stimulation failed to elicit either the excitatory or the inhibitory response. The height of the excitatory response to mechanical stimulation of the tooth was reduced after anesthesia of the tooth (Goldberg 1971, Sessle and Schmitt 1972) and the period of EMG inactivity was reversibly abolished (Sessle and Schmitt 1972).

The second period of EMG inactivity following electrical or mechanical stimulation of a tooth or other oral site has also received a number of different interpretations. This is in part attributable to the disagreement in the literature concerning under what stimulus conditions the response is obtained. Some experimenters have reported that they were able to obtain the response only at high levels of stimulation (Bratzlavasky 1972a, c, 1973; Yemm 1972a,b (using electrical stimulation); Bratzlavasky 1972c; Sessle and Greenwood 1976 (using mechanical stimulation)). On the other hand Yu, Schmitt and Sessle (1973) report obtaining the response with

innocuous stimuli. Bratzlavasky (1972c) reports that at the stimulus levels which were adequate to achieve the second period of EMG inactivity the first period of EMG inactivity was increased slightly in duration. Yemm's (1972a) results show a gradual decrease in the amplitude of the EMG at regular time intervals after the initial stimulus, decreasing in size with increase in the time from the stimulus. Sometimes a single, long-duration period of EMG inactivity is found at the stimulus levels adequate for achieving the second period of EMG inactivity, (Yu et al 1973) or, according ot Bratzlavasky (1972c) only at levels which are yet higher than the levels of stimulation required to elicit the second period of inactivity. Bratzlavasky (1972c) reports this single period of EMG inactivity to be 50-80 msec in duration. Bratzlavasky (1972a, c, 1973) suggests that the second period of EMG inactivity is nocioceptive and serves a protective function. Yu et al propose that it is caused by stimulation of high-threshold receptors with long, slowly-conducting pathways. Yemm (1972a, b) has suggested another explanation.

Yemm (1972a, b) has noted oscillations, or reductions in the amplitude of the EMG at regular time intervals after the initial stimulus. Although actual latencies of these reductions are not reported, from inspection of photographs of averaged responses presented by Yemm (1972a) it can be estimated that they occur at regular intervals of 35-40 msec, after the earliest period of EMG inactivity. No

response is found in digastric, even when the stimulus is painful (Yemm 1972b). Yemm (1972a, b) has investigated this phenomenon. By transducing jaw movements while stimulating the palate electrically he found that periods of EMG inactivity after the first period of EMG inactivity were time-locked to the jaw's opening subsequent to the first period of EMG inactivity (1972b). Jaw-opening started 8-10 msec after the beginning of the first period of EMG inactivity. He also found (1972a, b) a biphasic peak of EMG activity at the end of each period of EMG inactivity. He speculated (1972a) that this peak might be due either to the synchronization of activity of the muscle spindles in the masseter muscle following their simultaneous silence (as has been suggested to be the case for the peak of EMG activity found at the end of a silent period initiated in skeletal muscle (Alston, Angel, Fink and Hofmann 1967)) or it might be a stretch reflex initiated by jaw-opening movement caused by the period of EMG inactivity. He later found that jaw-opening started 5-10 msec before the peak of EMG activity concluding the period of EMG inactivity (1972b). Goldberg (1972a) found that the second phase of depolarization of masseter motoneurones following electrical stimulation of the lingual nerve of anesthetized cats (referred to above) disappeared when the cat's jaw was held in position. Investigating the possibility that the rubber bung placed between the subjects' teeth was contributing to the oscillation, Yemm replaced the rubber bung with a solid

block. He found the amplitude of the oscillation to be reduced (1972b). Yemm favors the conclusion that the second and subsequent periods of EMG inactivity are silent periods associated with autogenic stretch reflexes.

Yemm (1972a) has also pointed out that the latency of the first period of EMG inactivity following electrical stimulation of intraoral sites corresponds with the latency of the silent period following elicitation of a jaw-jerk reflex. It is not clear what significance, if any, he attaches to this fact since he also states that the biphasic synchronization of the EMG preceding the silent period is never present preceding the period of EMG inactivity. However, some of his photographs of averaged responses do seem to indicate the presence of a biphasic peak of activity. If the masseteric response is a true inhibitory response then it is an atypical jaw-opening reflex, since it is not accompanied by any activity in the digastric muscle (Yemm 1972b).

Anatomy of Oral Reflexes

All of the muscles of mastication except for the intrinsic lingual muscles, stylohyoid, geniohyoid and the posterior belly of digastric (which are supplied by the hypoglossal and facial nerves) are supplied by the trigeminal nerve. Thus the study of the physiology of mastication (and of speech) is largely the study of the

trigeminal nerve. The sensory root (the Gasserian ganglion) consists of three divisions; the ophthalmic, maxillary and mandibular divisions. The motor root (which contains proprioceptive afferents) joins the main nerve just distal to the Gasserian ganglion. The trigeminal complex includes several nuclei: the mesencephalic nucleus (MSV), which contains the cell bodies of the trigeminal muscle spindles, the cell bodies of some of the periodontal ligament receptors (Cooper, Daniel and Whitteridge 1953; Corbin and Harrison 1940; Jerge 1963a; Szentagothai 1948; Smith and Marcarian 1968), some of the cell bodies of masseteric Golgi tendon organs (Kawamura 1964a; Kawamura, Funakoshi and Tsukamoto 1958; Kawamura, Funakoshi and Takata 1960) and cell bodies of tongue tension receptors (Smith and Marcarian 1968); the sensory nucleus (sensory V), which contains cell bodies of trigeminal tactile and pain receptors and some of the cell bodies of periodontal receptors (Jerge 1964) and of masseteric Golgi tendon organs (Kawamura 1964a; Kawamura et al 1958, 1960); the motor nucleus (motor V), which contains the cell bodies of trigeminal alpha motoneurones; and the nucleus supratrigeminalis (STV), which consists of the cell bodies of interneurones (Jerge 1963b). The pathways involved in the reflex control of chewing include ones between various of these trigeminal nuclei, and between trigeminal and hypoglossal and facial nuclei.

The tongue is supplied by three nerves; the hypoglossal nerve supplies the motor efferents to the tongue (and some

claim that it also contains afferent fibers as well: this will be discussed shortly), the lingual nerve supplies the anterior two-thirds of the tongue with an afferent nerve supply, as well as serving as the afferent nerve from the lower teeth and gum (and some claim that it also carries muscle spindle afferents: this will also be discussed shortly), and the glossopharyngeal nerve, which is the afferent nerve supply for the posterior one-third of the tongue.

There have been conflicting results concerning the question of the presence of afferent fibers in the hypoglossal nerve. Tarkhan (1936b), Downman (1939) (using cats), Weddell et al (1940) (using rats) and Tarkhan and Abou-El-Naga (1947) have all reported the presence of sympathetic afferents in the hypoglossal nerve of cats. Tarkhan (1936a) (using rabbits) and Tarkhan and El-Malek (1950) (using rats, rabbits, cats, dogs and humans) have reported finding sensory cells located along the hypoglossal nerve which they believed to be proprioceptive cell bodies. Pearson (1943) found bipolar neurons in the hypoglossal nerve of human fetuses and proposed their function was probably sensory. Downman (1939), Green and Negishi (1963), Nakamura (1968) and Nakamura, Goldberg, Mizuno and Clemente (1970) have all proposed the existence of afferent fibers along the entire course of the hypoglossal nerve. Sauerland and Mizuno (1968) have reported obtaining polysynaptic discharges in the cervical vagus and recurrent laryngeal

nerves in response to stimulation of the proximal cut ends of the hypoglossal nerve. They contend that the afferents mediating this reflex leave the hypoglossal nerve at the nodose ganglion, join the vagus and enter the brain stem throught the jugular foramen. Hanson and Widen (1970) have found that stimulation of high threshold afferent fibers following the same course elicits bilateral twitching of the vibrissae and suppression of shivering in cats. Morimoto and Kawamura (1971, 1972) and Kawamura and Morimoto (1973) have found that high-threshold afferents in the hypoglossal nerve of cats (primarily the lateral branch, which innervates the retractive muscles) produce IPSP's in the motoneurones contained in the medial branch of the hypogolssal nerve. Carleton (1938) (using rabbits), Weddell et al (1940) (using rats and rabbits), Boyd (1937, 1941) (using rabbits) and Law (1954) (using infant pigs) have disconfirmed the presence of hypoglossal sensory cells and/or ganglion cells located along the nerve and Barron (1936) (using cats, rats and rabbits), Corbin and Harrison (1938b) (using cats), Blom (1960) (using cats) and Porter (1966a) (using cats) have reported failing to elicit any impulses in the hypoglossal nerve in response to a number of different stimuli applied to the tongue. Observations by Langworthy (1924a, (using cats), Van der Sprenkel (1934) (using hedgehog) and Corbin, Lhamon and Petit (1937) (using monkey) suggest that the upper cervical dorsal root ganglia contribute proprioceptive afferents to the hypoglossal

nerve. However, Hinsey and Corbin (1934) found no myelinated fiber degeneration in the peripheral portion of the hypoglossal nerve of the cat after removing the upper four cervical dorsal root ganglia and Corbin and Harrison (1938b) obtained similar results after sectioning of the first two cervical nerves of cats. Cooper (1954) and Blom (1960) have reported finding afferent impulses in filaments of the medial end-branch of the hypoglossal nerve in cats in response to stretching of the tongue. Porter (1966a) has criticized Cooper's finding, pointing out that since Cooper's study was performed four years prior to the description by Fitzgerald and Law (1958) (confirmed by Blom (1960)) of a peripheral anastomosis between the lingual and hypoglossal nerves, Cooper may have in fact been recording lingual nerve potentials. Both Cooper and Blom have acknowledged this possibility and Blom (1960) has reported failing to obtain afferent impulses while recording from the main trunk of the hypoglossal nerve. Hanson and Widen (1970) have recorded afferent impulses in the proximal part of the main trunk of the hypoglossal nerve of cats in response to stretching of the tongue. Nakamura et al (1970) have found afferent impulses in the hypoglossal nerve of cats, ascending polysynaptically and bilaterally via the hypoglossal roots and motor V which inhibit (at a latency of 8-11 msec) the masseteric monosynaptic reflex, followed by a late (latency 40-80 msec) period of suppression). Zapata and Torrealba (1971) have reported the presence of slowly-

adapting sensory nerve fibers in the hypoglossal nerve of the cat which responded to longitudinal or lateral tensions or displacements of the tongue but not to tactile stimuli applied to the surface of the tongue. Most units they studied showed a basal discharge rate at rest. Their rate of discharge during mechanical stimulation depended on the intensity of the stimulus and its acceleration during application or removal. Those units which showed base-line activity also showed a short pause in discharge when the simulus was removed abruptly. The available evidence would thus seem to favor the view that for non-primates there probably are sympathetic and nocioceptive afferent fibers present in the hypoglossal nerve and there possibly are proprioceptive afferent fibers present in the nerve as well.

There is conflicting evidence concerning the situation for primates as well. Bowman and Combs (1969) and others have presented evidence that for macaca mulatta the lingual muscle spindle afferents travel in the hypoglossal nerve and branch off there to enter the central nervous system by the cervical dorsal roots. While Bowman suggests that these results may well generalize to all primates the fact is that the evidence we have for humans is quite indirect. Adatia and Gehring (1972), for example, arguing that bilateral lingual nerve blocks are a safe procedure to use in dental practice, point to the fact that patients with bilateral lingual nerve blocks have no difficulty producing even the most complex utterances and conclude that lingual muscle

spindle afferents cannot be in the lingual nerve (therefore bilateral lingual nerve blocks are a safe procedure). Bratzlavasky (1972b) found that brisk tongue tap evoked an uncrossed excitatory reflex response in the orbicularis oris muscle of humans with a latency of 11-15 msec. His photos show a second excitatory response occurring at approximately a 80 msec latency, but he did not comment on this. The response was unaffected by surface anesthesia but was abolished by lingual nerve anesthesia. It was facilitated by orbicularis oris contraction. Since its latency is greater than that of the jaw-jerk reflex Bratzlavasky comments that "it appears to have a polysynaptic pathway." In a later discussion (1973) when discussing the fact that the reflex is not evoked by stretching of the tongue, he says

> Electrical supramaximal stimulation of the hypoglosssal nerve excites all afferent fibers simultaneously which may result in antagonistic effects between IA and other afferents, masking possible reflex responses. The same could occur upon stretching of the tongue musculature, in view of the tridimensional arrangement of the muscle fibers and thus of the muscle receptors in this organ.

Some additional anatomical facts, which apply equally to non-primates and primates, have been gathered as well, the results of which are not particularly suggestive of one pathway or the other. The results include the following: (1) the hypoglossal nerve contains no sensory root, which seemingly all (other?) sensory nerves have and (2) neither

the hypoglossal nerve nor the lingual nerve has the caliber spectrum which is typical of nerves which contain muscle spindle afferents; the large diameter nerve fibers are far fewer in relative number in these nerves than they are in typical spinal nerves (Blom 1960; Egel, Bowman and Combs 1968). Neither of these results is conclusive, of course, because cranial nerves may not correspond to generalizations which can be expressed concerning spinal nerves. With respect to the former point it will be recalled that some (Tarkhan 1936a; Tarkhan and El-Malek 1950) have claimed that in both humans and non-humans the cell bodies of lingual proprioceptive afferents are found along the hypoglossal nerve, in opposition to the normal location in a sensory root, but these results have been disputed (Boyd 1937, 1941; Carleton 1938; Weddell et al 1940; Law 1954). And with respect to the latter point it may be mentioned that efferent fibers in the masseteric nerve are smaller than the corresponding types of fibers in spinal nerves: in contrast with the spinal IA fiber diameter range of 13 to 22 microns, the largest fibers in the masseteric nerve are 12 microns in diameter. Furthermore only one percent of the fibers in the masseteric nerve are of that diameter (Smith, Dale and Marcarian 1968).

In fact a large number of studies, dating from before it was known that non-primates do not possess lingual muscle spindles, have shown that the lingual nerve of non-primates contains spindle-like afferents. In fact, as would be

expected, the earlier studies on this topic used the finding of spindle-like response patterns in the lingual nerve as evidence of the presence of muscle spindles in the tongues of animals like the cat. This was part of the reason why the debate over the question of the presence of muscle spindles in the tongue persisted for such a long time. Miller and Sherrington (1915) reported producing twitching of the tongue by stimulating the lingual nerve of cats. It has been shown that lingual nerve afferents in cats synapse di-synaptically with the ipsilateral hypoglossal nerve (Porter 1965, 1967; Sumino and Nakamura 1973) and tri-synaptically with the contralateral hypoglossal nerve (Porter 1965, 1967) and that lingual nerve stimulation can produce excitatory post-synaptic potentials (EPSP's) (Blom 1960; Porter 1965), IPSP's (Biedenback and Chan 1971; Morimoto and Kawamura 1972; Morimoto *et al* 1968, 1972; Sumino and Nakamura 1973) or EPSP-IPSP sequences (Porter 1967; Morimoto *et al* 1968; Morimoto, Takata and Kawamura 1972; Sumino and Nakamura 1973) in hypoglossal motoneurones. The afferent fibers involved in this reflex conduct nervous impulses at a speed of 48 m/sec and the efferent fibers conduct at a speed of 36-48 m/sec (Blom 1960). Some have found that lingual nerve stimulation (Morimoto *et al* 1968) or mechanical stretch of the tongue (Kawamura, Funakoshi, Nishiyama and Morimoto 1967) produces reciprocal effects in tongue retractive and protrusive motoneurones. Others (Sumino and Nakamura 1973) have found similar effects in

both types of motoneurones, in response to electrical stimulation of the lingual nerve. Kawamura et al (1967) found that the responses due to stretch of the tongue were not abolished by surface anesthesia of the tongue but were abolished by lingual nerve section. Law (1954) has described a non-muscle spindle type of muscle receptor present in the tongues of the cat, dog and pig. She found coiled nerve terminals situated within an oval or pear-shaped capsule, in the intra-muscular connective tissue of the tongue, the long axis of which was parallel to the muscle fibers. These structures were most abundant in the intrinsic muscles of the tongue, between the vertical and transverse muscles. A few were found in the inferior longitudinal muscle and none were found in the superior longitudinal muscle. Owing to its structure and location this receptor would seem to be capable of initiating proprioceptive afferent impulses. A similar type of receptor has been found at the base of the genioglossus muscle at the point of its attachment to the mandible (Weddell et al 1940). Two types of lingual mechanoreceptors have been isolated physiologically; superficially-situated rapidly-adapting receptors with fast nerve conduction velocities (40 m/sec average) and deeply-situated slowly-adapting receptors with slower nerve conduction velocites. These receptors have been found to respond to stretching of the lingual musculature (Porter 1966a). Hensel and Zotterman (1951) have estimated the mechanoreceptor fibers

to be 8-10 and 12-15 microns in diameter. Nakamura (1971) has measured the conduction velocity of lingual nerve fibers in cat as 50-66 m/sec by stimulating the spinal nucleus of the trigeminal nerve and recording the antidromic spikes in the lingual nerve. Thus it seems that although non-primates have no lingual muscle spindles, they do have lingual mechanoreceptors which behave like muscle spindles. Furthermore, the majority of the literature favors the view that for non-primates the afferent fibers from mechanoreceptor afferent fibers travel in the lingual nerve.

CHAPTER II

PROCEDURES

The experiments were performed on a total of six subjects; three male and three female. Ages ranged from twenty to twenty-eight years.

Subjects were seated upright in a comfortable chair. The experimental procedure was explained to them. The attending clinician obtained the subjects' medical history. Bipolar surface silver disc electrodes were placed on the skin over each of the subject's masseter muscles and retained by adhesive discs. Correct placement was determined by palpation of the contracted masseter muscle. Electrodes were placed over the center of the muscle, parallel to the direction of the muscle fibers. A ground electrode was placed on the back of the neck.

The EMG traces were amplified by means of two differential amplifiers. Right masseter EMG was amplified by a Tektronix 3A9 differential amplifier (frequency response 0.1 KHz to 10 KHz). Left masseter EMG was amplified by a Tektronix 2A61 differential amplifier (frequency response 60 Hz to 100 KHz). The oscillcscope and stimulator were triggered by a Devices digitimer 3290. The oscilloscope was triggered 10 msec before the stimulator. Stimulus pulses were delivered at a rate of one per second. A DC square pulse of 0.2 msec duration was produced by a Devices Mk IV isolated stimulator and delivered by bipolar

stainless steel wire electrodes (interpolar distance approximately 4 mm) held in position by the experimenter. The EMG traces and stimulus pulse were monitored by means of a Tektronix 3A74 four-trace storage oscilloscope. The EMG traces and the pulse used to trigger the oscilloscope were recorded on tape by means of a Thermionic four-channel tape recorder. Stimulus voltage levels were recorded on the fourth channel of the recorder through a microphone. Averaging (integration) of series of responses was accomplished by means of a Biomac 1000 usually after the signal had been rectified. Oscilloscopic traces and averaged signals were photographed by means of a Tektronix oscilloscope camera C-2.

Subjects were first trained to maintain a steady clench on two rubber bungs placed between his upper and lower back teeth. Gain on the amplifiers was adjusted so that sufficient signal was obtained while the subject was clenching at a pressure which he felt would not be uncomfortable after a prolonged time. The stimulating electrodes were placed lateral to the mid-line of the dorsum of the tongue. Prior to the application of anesthesia various thresholds were determined; threshold for sensation of the stimulating pulse, threshold for the subject's reporting a change in the subjective quality of the stimulus, and threshold for obtaining periods of EMG inactivity. Approximately 40 recordings were made at each of several voltage levels, usually starting at the voltage

level of the threshold of sensation and raising the voltage beyond the threshold for obtaining periods of EMG inactivity.

The lingual nerve was unilaterally anesthetized by a clinician, central to its anastomosis with the hypoglossal nerve. In no case was the muscular control of the tongue lost. 2% Xylocaine with epinephrine was the anesthetic used. Except in one case anesthesia was applied to the right lingual nerve. After it was determined that the anesthesia had taken effect (using von Frey's bristles), the same thresholds as previously determined were again determined while stimulating on the unanesthetized half of the subject's tongue. Stimulation was then applied to the anesthetized half of the tongue, again starting at the subject's pre-anesthesia threshold for sensation and gradually increasing the voltage to up to 3 times the pre-anesthesia threshold for obtaining silent periods.

For some subjects two experimental sessions were used; one pre-anesthesia session and one post-anesthesia session. Pre-anesthesia thresholds were determined immediately before anesthesia in all cases.

Some additional procedures were followed for two subjects, one of whom received bilateral lingual nerve block anesthesia. A patella hammer was used to apply a tap to the jaw and to the tongue of the subject and to simultaneously trigger the scope. The jaw tap procedure was the same as that used by other experimenters (Goldberg 1971). The

tongue tap procedure involved the experimenter's grasping the subject's tongue firmly and tapping his own thumb nail (located over the dorsum of the subject's tongue) with the head of the patella hammer. The subject was instructed to relax his jaw-closer muscles. These procedures were carried out both before and after lingual nerve block anesthesia. Also, the effect of having the subject clench on a gauze pad instead of on rubber bungs was determined with these same 2 subjects.

In three experimental sessions surface anesthesia of the tongue using a spray application of orange-flavored 2% Xylocaine was examined for its potential usefulness as an experimental technique. Pre-test touch and pressure thresholds were determined. Xylocaine was sprayed on the surface of the tongue while the experimenter held the subject's tongue pulled out of the subject's mouth. An assistant held cardboard over the subject's lips in an attempt to prevent the lips from being anesthetized. Touch and pressure thresholds were again determined. Informal observations of motor control of speech were made while conversing with the subject. A number of re-applications of anesthetic were done. No attempts were made to monitor the amount of anesthetic used. With one subject the threshold for achieving consistent periods of EMG inactivity was determined before and after anesthesia. Subjects were asked for a subjective account of the experience.

CHAPTER III

RESULTS

PRE-ANESTHESIA

The threshold for sensation of the stimulus pulse voltage varied between subjects from 2 to 7 volts (see Table 1). The threshold also varied within the same subject, from the first session to the second, the differences in threshold voltages being from 0.5 to 3 volts. The mean threshold for sensation (averaging over all subjects and all sessions) was 4.2 volts.

TABLE 1. THRESHOLD OF STIMULUS PULSE

Subject	Threshold (in volts)	
	Session 1	Session 2
1	4	
2	4-1/2	6-1/2
3	4	7
4	3	3-1/2
5	2	3-1/2
6	4	

All subjects reported that the quality of the stimulus changed from being a "tap" to being a "pinch" or "tingle" as the voltage of the stimulus was increased. Most subjects reported the change occurring below 23 volts (see Table 2). For all subjects periods of EMG inactivity were found at a number of different latencies. In order to distinguish them, the period of EMG inactivity appearing at the shortest

latency will be termed "the first period of EMG inactivity", the one appearing at the second shortest latency will be termed "the second period of EMG inactivity", and so on.

TABLE 2. THRESHOLD OF CHANGE IN SUBJECTIVE STIMULUS QUALITY

Subject	Threshold (in volts) Session 1	Session 2
1	19	
2	20	19
3	18-21	19-21
4	21	20
5	18-21	20-21
6	23	

The first period of EMG inactivity had a latency of 11 to 20 msec. The mean latency was 16 msec (n=138, s.d.=3.00 msec)). Its duration was 8 to 22 msec, and occasionally (3 occurrences in 104) it was much longer (35-40 msec), these longer duration pericds of EMG inactivity always corresponding to the absence of a second period of EMG inactivity. The mean duration was 14 msec (n=104, s.d.=4.57).

The second period of EMG inactivity had a latency of 40 msec to 54 msec. The mean latency was 44 msec (n=76, s.d.=3.2). Its duration was 6 msec to 15 msec. The mean duration was 10 msec (n=76, s.d.=2.5).

The first pericd of EMG inactivity appeared more consistently (i.e., in a higher percentage of trials at the same voltage level) than the second period of EMG

inactivity. In 106 consecutive trials at the same voltage level there were 104 first periods of EMG inactivity (i.e., first periods of EMG inactivity appeared in 98.1 per cent of the trials) whereas there were 76 second periods of EMG inactivity (i.e., second periods of EMG inactivity appeared in 71.7 per cent of the trials).

Either or both of the first and second periods of EMG inactivity would occasionally occur (i.e., they would appear in some trials and not appear in other trials) at voltage levels as low as 18 to 20 volts. The presence of periods of EMG inactivity was sometimes associated with the presence of a visible local twitch or contraction of the tongue muscles. The percentage of trials in which the periods of EMG inactivity occurred would continue to increase as the voltage was increased, up to a certain voltage level and then would no longer increase. The "threshold for achieving consistent periods of EMG inactivity" was defined as the lowest voltage level at which the percentage of trials in which periods of EMG inactivity were present was maximal, or in other words, the voltage level above which the percentage of trials in which periods of EMG inactivity appeared would not increase. The threshold for achieving consistent periods of EMG inactivity varied between subjects from 24 volts to 30 volts, the mean threshold being 27.2 volts (see Table 3). In most cases the first and second periods of EMG inactivity would begin to appear at about the same voltage level, within the same subject, and the threshold for

TABLE 3. THRESHOLD OF ACHIEVING CONSISTENT SILENT PERIODS

Subject	Threshold (in volts) Session 1	Session 2
1	26-27	
2	26	28
3	30	27-28
4	26	27
5	24-25	30
6	27	

achieving the first period of EMG inactivity consistently was the same as the threshold for achieving the second period of EMG inactivity consistently. For two subjects, though, the threshold for achieving the second period of EMG inactivity consistently was at a lower voltage level than the voltage level associated with the threshold for the consistent occurrence of the first period of EMG inactivity. Two sessions were used for each of these subjects. In both cases different results (results which were consistent with those obtained for other subjects) were obtained in the second session.

As many as 6 or 7 periods of EMG inactivity may be clearly distinguished in the averaged EMG. Those periods of EMG inactivity appearing after the second period of EMG inactivity were not measured for individual trials. However, the latencies of these later periods of EMG inactivity can be estimated by looking at averagings such as shown in Figure 1. They are shown to appear at fairly regular intervals of approximately 27 msec. Furthermore, the "depth" of each period of EMG inactivity is seen to be

reduced with respect to the period of EMG inactivity preceding it (in time). (I.e., the distance of the period of EMG inactivity from the baseline is greater for each successive period of EMG inactivity.) All periods of EMG inactivity except for the first and second ones disappeared when the subject was required to clench on a gauze pad instead of on rubber bungs. (Figure 2).

POST NERVE-BLOCK ANESTHESIA

All periods of EMG inactivity disappeared after unilateral lingual nerve block, when the stimulating electrode was placed on the anesthetized side of the tongue. The same result was obtained for all subjects. Local twitch contractions were still present. It was observed that occasionally a period of EMG inactivity would appear in the EMG trace when stimulating on the anesthetized side of the tongue but not often enough that these periods were present in the averaged EMG. These periods of EMG inactivity were always seen to correlate with the local twitch crossing the midline of the tongue. These periods of EMG inactivity were never seen when the local twitch did not cross the midline of the tongue.

All of the periods of EMG inactivity present in the EMG before anesthesia were still present when the stimulating electrodes were placed on the unanesthetized side of the tongue after lingual nerve block anesthesia.

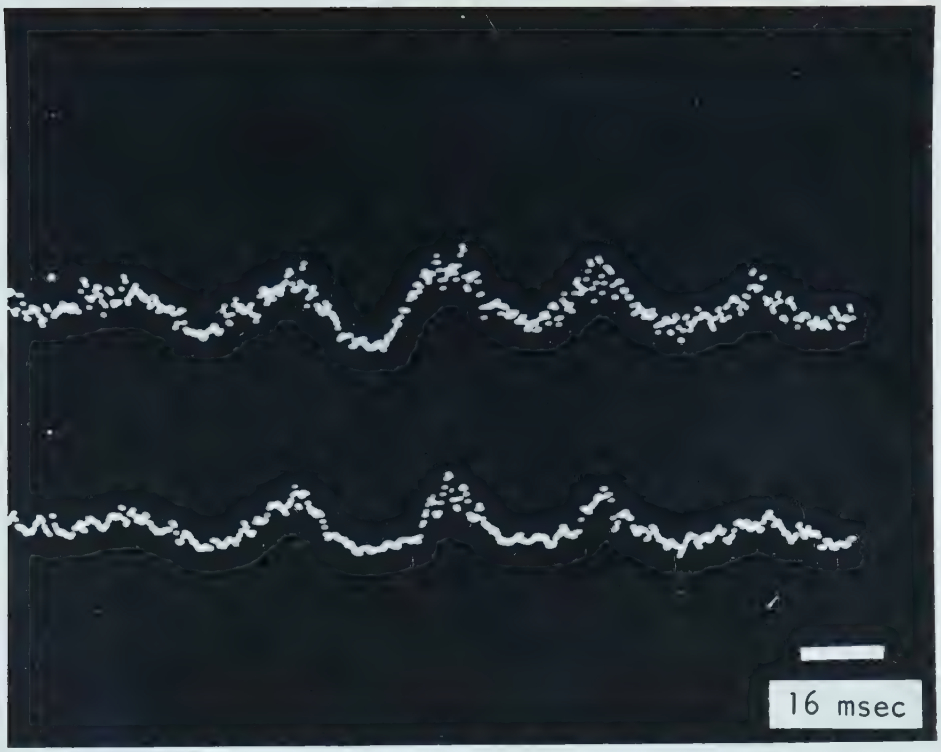

Figure 1: Electrical Stimulation of left side of Tongue. Subject clenching on Rubber Bung. Top Trace = Left Masseter EMG. Bottom Trace = Right Masseter EMG. n=64. Peaks at 19,46,74,97,128 msec.

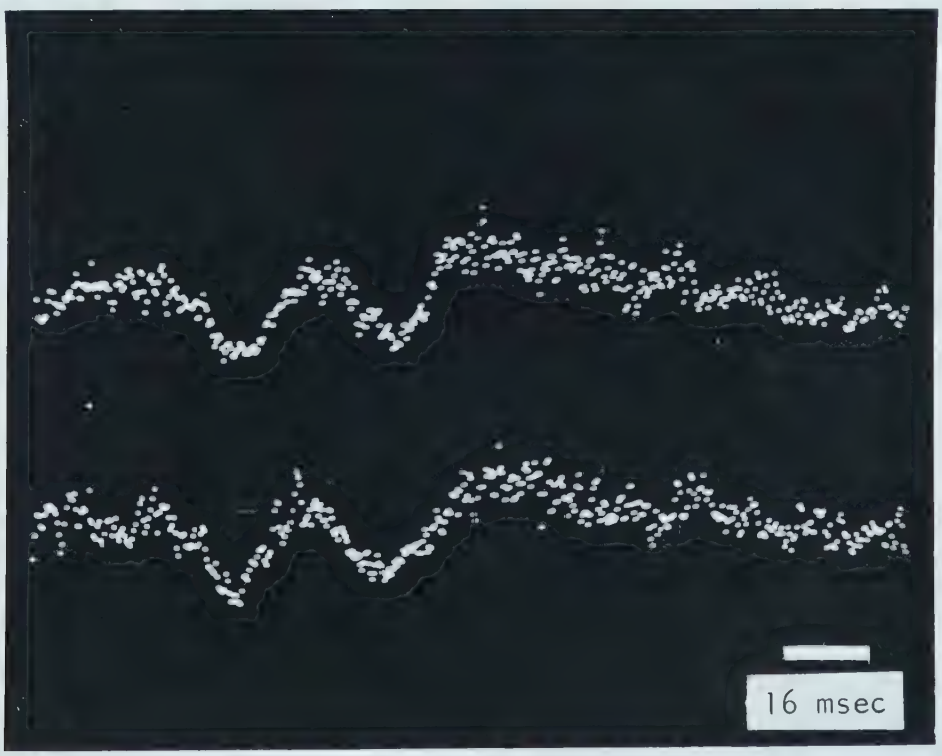

Figure 2: Electrical Stimulation of left side of Tongue. Subject clenching on gauze. Top Trace = Left Masseter EMG. Bottom Trace = Right Masseter EMG. n=64. Peaks at 24 and 40 msec.

Jaw-Jerk

The jaw-jerk reflex was easily evoked using the procedures outlined in Chapter II (application of a tap to the jaw of the subject using the patella hammer). A photograph of 32 rectified and averaged jaw-jerks is shown in Figure 3. The mean latency of the jaw-jerk was 8.0 msec (n=68, s.d.=0.03 msec). Its mean duration was 6.2 msec (n=68, s.d.=0.61 msec). The jaw-jerk reflex was still present after bilateral lingual nerve block anesthesia.

Tongue-Jaw Jerk

A tongue-jaw jerk reflex could be evoked using the procedures outlined in Chapter II (application of a tap to the tongue of the subject using the patella hammer). However, the tongue-jaw jerk was not easily evoked. In fact it did not appear consistently enough to do an averaging. The tongue-jaw jerk was found to be approximately 2 msec longer in latency than the jaw-jerk (Figure 4). It was not possible to obtain the tongue-jaw jerk after bilateral lingual nerve block anesthesia. Because the tongue-jaw jerk is not easily evoked many trials were attempted, but never was a tongue-jaw jerk obtained.

POST SURFACE ANESTHESIA

All subjects reported difficulty in speaking. All reported that their tongue felt thick and unwieldy; that they had to "push it" around their mouth. Two subjects'

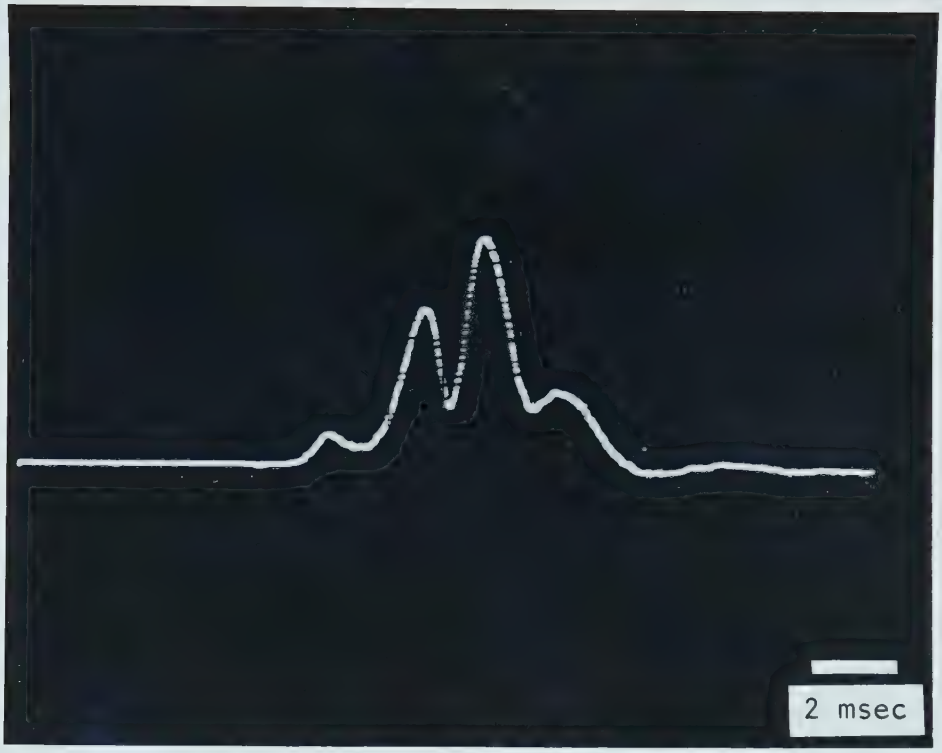

Figure 3: Jaw-Jerk, n=32. Oscilloscope Triggered by Patella Hammer.

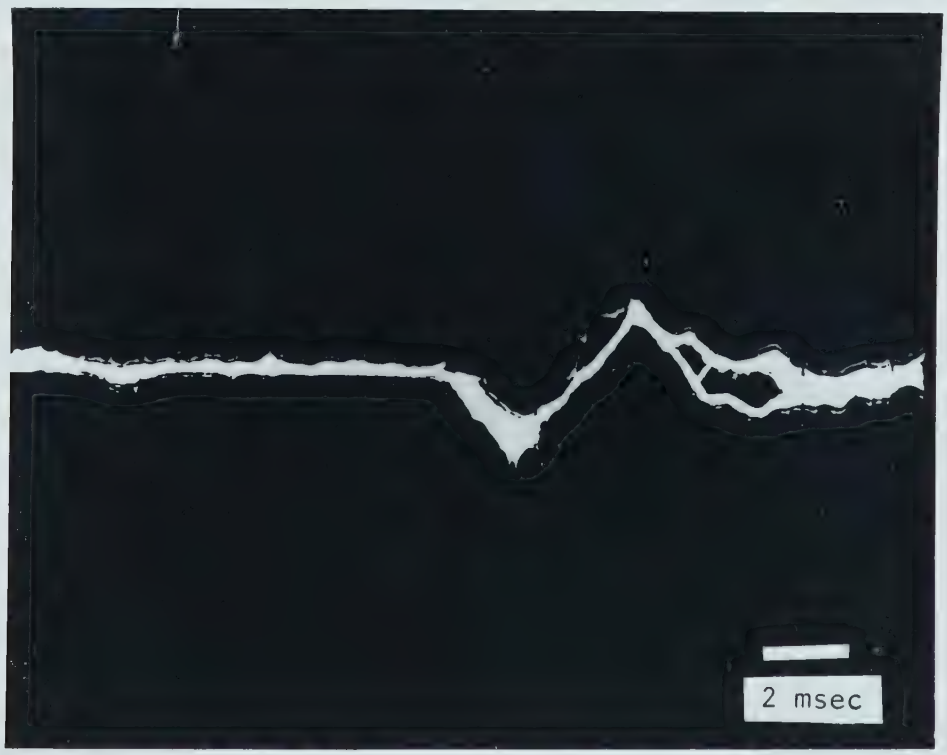

Figure 4: Tongue-Jaw Jerk, n=1. Oscilloscope Triggered by Patella Hammer.

speech definitely seemed to the experimenters to be slurred. One subject reported that her voice sounded "far away", like it was not her own voice. Touch and pressure sensation were reduced to varying degrees. Neither was eliminated completely, but touch was always reduced at a lower anesthetic level than pressure was. One subject reported partial and one reported total loss of sensation in his lips. All reported partial loss of sensation in the hard palate. Two reported apparent loss of sensation in the pharynx and some difficulty with swallowing. All subjects salivated more than normally. Masseteric periods of inactivity were never eliminated, although the threshold for achieving them was raised slightly.

CHAPTER IV

DISCUSSION

ELECTRICAL STIMULATION

As stated in Chapter I, in order to use the "silent period technique" it is necessary to show the presence of a monosynaptic excitatory reflex in the same or in synergistic muscle fibers. Without this excitatory response a period of EMG inactivity cannot be identified as a silent period. In this study it was not possible to clearly identify such an excitatory response in masseter muscle EMG preceding the observed period of EMG inactivity. The following conclusions can be drawn from the electrical stimulation experiments:

(1) It is unlikely that pain receptor afferents are involved in producing the periods of EMG inactivity. No subjects reported the stimulus to be painful.

(2) A twitch-sensitive receptor is implicated. This conclusion is drawn from the observation that periods of EMG inactivity were found in the contralateral masseter muscle EMG after lingual nerve anesthesia when stimulating on the anesthetized side of the tongue in all and only those cases in which extrafusal muscle fiber contraction was observed to cross the mid-line of the tongue.

(3) Whichever afferent fibers mediate the observed periods of EMG inactivity, they travel in the lingual nerve, at least past the anastomosis with the hypoglossal nerve.

This conclusion is drawn from the observation that these periods of EMG inactivity were not present following anesthesia of the lingual nerve.

(4) Receptors located on the surface of the tongue are probably not responsible for the periods of EMG inactivity. Otherwise the periods of EMG inactivity would have been eliminated following surface anesthesia of the tongue.

(5) The third, fourth, etc. periods of EMG inactivity were most likely due to bounce. They were present when rubber bungs were placed between the subjects' teeth but were eliminated or reduced when gauze was placed between the subjects' teeth.

MECHANICAL STIMULATION

Examination of the difference between the latency of the tongue-jaw jerk reflex and the latency of the jaw-jerk reflex establishes the monosynaptic nature of the tongue-jaw jerk reflex. The latency components of each are the following: (1) receptor delay (0.2-0.3 msec (Lloyd 1943); .5 msec (Jerge 1963a)), (2) afferent transmission time, (3) synaptic delay (0.5-1.36 msec (Lorente de No 1935);0.5-.9 msec (Renshaw 1940)), (4) efferent transmission time and (5) neuromuscular delay (0.7 msec (Lorente de No 1935); 0.55 msec (Eccles and O'Connor 1939; Lloyd 1941)). The difference between the latency of the tongue-jaw jerk and the latency of the jaw-jerk reflex is the difference in afferent conduction time. The afferent pathway for the jaw-

jerk reflex is approximately 81 mm in length.[8] The lingual nerve is approximately 200 mm in length.[9] Unfortunately no direct measures of the conduction velocities of the appropriate nerves have been performed. Thus an approximation must be used. Two related sorts of data may be used if forming an approximation; the speed of conduction of other IA fibers in non-humans and humans and the diameters of the largest fibers in the lingual and masseteric nerves in man.

The conduction velocity of spinal IA fibers in the cat is 70-120 m/sec (Ganong 1975; Lloyd 1943). Conduction velocities of terminal endbranches of nerves are lower, owing to narrowing of nerve fiber diameters (Buchthal and Rosenfalck 1966). Furthermore, cranial nerves in general may have smaller nerve fiber diameters than the corresponding spinal nerves have. As mentioned above (Smith et al 1968) this is the case at least for the fibers of the masseteric nerve. Whereas the alpha fibers from the gastrocnemius nerve 8 mm from the muscle range in diameter from 11 to 15 microns (and thus conduct at between 66 and 90 m/sec (Ruch et al 1965) the speed of conduction of the masseteric alphas in cats is approximately 39 m/sec (Sessle and Greenwood 1976). This (latter) value is comparable with values found for facial, ambiguous and hypoglossal neurones (Sessle and Greenwood 1976).

Calculating from cur own measure of the latency of the jaw-jerk reflex the speed of conduction along the fibers is

24-33 m/sec. However, an unknown amount of time is attributable to delay between time of triggering (by the patella hammer) and arrival of the stimulus at the receptors, so these values are underestimations. Fujii and Mitani (1973), using electrical stimulation of the masseter muscle, found the latency of the H wave of the masseteric reflex in humans tc be 6.0 msec. Bratzlavasky (1972a, c) found the same result in a similar experiment. Estimating the distance from their stimulating electrodes to the center of the nerve to be 110 mm (the estimate made by Fujii and Mitani) and allowing 1.0 msec for intermuscular delay, the average conduction velccity of the nerves involved is calculated to be 67 m/sec.

The difference between the latency of the jaw-jerk reflex and the tongue-jaw jerk reflex was 2.0 msec. Using the nerve conduction velocity estimate of 70 m/sec (which seems to be the most reasonable estimate to use) the difference between the afferent conduction time for the tongue-jaw jerk reflex and the afferent conduction time for the jaw jerk-reflex is 1.7 msec. Since the remaining time (0.3 msec) is insufficient for a second synapse the tongue-jaw jerk reflex must be a monosynaptic reflex, like the jaw-jerk reflex. The fact that this reflex could not be elicited after bilateral lingual nerve block is strong evidence that the afferent portion of the reflex is contained in the lingual nerve. That is, if the afferents mediating this reflex were located in the hypoglossal nerve

then the reflex would still appear after the lingual nerve block. The tongue-jaw jerk reflex could not have been confused with the jar reflex (Sherrington 1898; Hannam et al 1970) because of the fact that the tongue-jaw jerk reflex appeared at a different latency than did the jaw-jerk reflex whereas the jar reflex occurs at the same latency as the jaw-jerk reflex (Hannam et al 1970).

Of course the possibility cannot be overlooked that there are mechanoreceptor afferents in both the lingual and hypoglossal nerves. The evidence is very strong in the case of the cat that receptors other than muscle spindle receptors are involved in mechanoreceptor-initiated monosynaptic reflexes. It is not difficult to envision that receptors like those operating in the cat may also be in operation in humans, in addition to muscle spindles, especially in light of the similarity between the results from experiments on humans and results from experiments on non-humans. Considerable evidence suggests that afferent fibers mediating this reflex in cats are contained in the lingual nerve, and there is some evidence that mechanoreceptor afferents travel in the hypoglossal nerve of cats as well. In the case of primates we cannot ignore the evidence presented by Bowman and Combs, and Bowman in favor of the presence of lingual muscle spindle afferents in the hypoglossal nerve of the rhesus monkey, in spite of the problems that such a conclusion presents (most of which are acknowledged by Bowman). Although the situation for humans

cannot be assumed to be the same as the situation for rhesus monkeys, a priori we would expect that it was likely to be the same. Thus we are lead, by consideration of reflexes in non-humans, to the possibility that humans, like cats, may have mechanoreceptor afferents in the lingual nerve and, like rhesus monkeys, may have muscle spindle afferents in the hypoglossal nerve. One piece of evidence in this investigation suggests that if there is more than one kind of mechanoreceptor present in humans which is capable of mediating monosynaptic reflexes they both (all) travel in the lingual nerve. That is the evidence that when the lingual nerve was bilaterally anesthetized the tongue-jaw jerk reflex disappeared. If an appropriate set of afferents was contained in the hypoglossal nerve then the reflex should have persisted.

Although it should not be assumed that the receptor which initiated the tongue-jaw jerk reflex following mechanical stimulation was the same as the receptor which initiated the periods of EMG inactivity following electrical stimulation it may be concluded that the afferents involved in the production of each of these responses travel via the lingual nerve. The evidence for this conclusion is the fact that each of these responses disappeared following lingual nerve anesthesia.

SURFACE ANESTHESIA

Because we were unsuccessful in identifying an

excitatory monosynaptic response in the masseter muscle when electrically stimulating the tongue we do not know whether lingual muscle spindle activity was disrupted by the application of surface anesthesia; if the periods of EMG inactivity were silent periods then surface anesthesia is ineffective in disrupting muscle spindle functioning. There were other difficulties with the surface anesthesia technique which are worthwhile mentioning, however, since surface anesthesia has been used for purposes other than disrupting muscle spindle feedback. These include the following: (1) it is difficult to control the amount of area to be anesthetized, (2) it is difficult to determine and to control the level of anesthetization obtained, (3) the anesthetic effects are extremely short-lived,[10] (4) the technique causes a certain amount of discomfort in the subject; the subject can hardly avoid swallowing the anesthetic, which may cause anesthesia of the pharynx and mild discomfort in swallowing, often followed by mild stomach upset, (5) the effects seem to be far from specific, aside from the already-mentioned difficulties. The subject inevitably inhales some of the anesthetic. This can, of course, result in general anesthetic effects, and in fact subjects' reports of the effects of the anesthetic were consistent with that idea. It was also quite unclear what receptors might have been affected by the anesthetic. It appeared as though no sensory modality was completely eliminated, and that all (plus the alpha and gamma

motoneurones) may well have been affected to some extent. In conclusion, it seems that the use of surface anesthesia is not a potentially profitable technique in speech research.

SELECTIVE ANESTHESIA

Selective anesthesia involves anesthetization of some, not all, of the nerve fibers in a nerve. There have been two types of selective anesthesia techniques used.[11] Both depend crucially on the different effects of anesthesia upon nerve fibers of different diameters.

Leksell (1945) showed gamma motor fibers to be more sensitive to local anesthetics than large diameter motor fibers. In a series of articles Matthews and Rushworth showed that local anesthetics affect small diameter gamma fibers before they affect large diameter motor and afferent fibers and demonstrated the effects of selective anesthesia on muscle reflexes (Matthews 1956, Matthews and Rushworth 1956, 1957a, b, 1958). They found, for example, that muscle reflexes are not abolished until the large diameter motor fibers are paralyzed but rather are modified or reduced (Matthews and Rushworth 1957a). They proposed two explanations for the fact that these reflexes are modified or reduced: (1) loss of gamma tone causing the (flaccid) intrafusal fibers to be less sensitive to external stretch on the muscle, and (2) reduction of the excitability of the motoneurones brought about by the decrease in the resting

discharge from the muscle spindles. They provide support for each explanation (Matthews and Rushworth 1957b). Nathan and Sears (1964) found that "absolute differential blocking" is possible. In the previous studies reported here the small-diameter nerve fibers were said to be anesthetized before (temporally) the large-diameter fibers. Nathan and Sears were able to achieve small-diameter fiber blocks in which the large-diameter fibers were never anesthetized. They do not know whether to attribute their ability to achieve absolute differential blocks to the fact that they applied the anesthetic directly to the spinal roots or to the fact that they used lower levels of the anesthetic than were used in previous experiments.

Stark (1968), Smith (1969) and Abbs (1973) have performed experiments which utilized the principle that nerve fibers of large diameter recover more rapidly from the effects of anesthesia than do nerve fibers of small diameter. The Abbs experiment (the one in which masseteric nerve blocks were performed) was referred to already (in Chapter I).

CHAPTER 5

CONCLUSIONS

The unsettled question of the pathway of afferent nerve fibers from muscle spindles in the tongue of human subjects was resolved using a hitherto unused technique. Reflex connections between these nerve fibers and the masseter muscle were established and then disrupted by nerve anesthesia. That the reflex response to muscle spindle stimulation was abolished after lingual nerve anesthesia established that, in humans, the fibers from these muscle spindles travel in the lingual nerve. Two different types of muscle spindle stimulation were used, electrical and mechanical. The electrical stmulation was found to be inadequate to produce unambiguous results, but the mechanical stimulation clearly produced a monosynaptic response, which unambiguously identified muscle spindles. Techniques for blocking muscle spindle feedback from the tongue were discussed.

FOOTNOTES

1. The assumption being made here is that the place to start in developing a model of muscle spindle involvement in speech motor control is with the question of whether the models of motor control developed on the basis of evidence other than evidence concerning the human speech muscles, can be extended without modification to describing motor control of the human speech muscles. In fact it seems clear that we cannot do so; there are structural and functional differences between the systems which have formed the basis of the generation of motor control models, and the speech musculature, which make this impossible. Bowman (1971; 4) has stated this point in a particularly succinct fashion.

> It is, of course, apparent that certain recurring patterns of neural organization are found within the nervous system, suggesting that information about the structural and operational characteristics of the spindle system in the speech musculature may be gleaned from the study of the same system in the limb and trunk musculature. Such extrapolation clearly assumes that the spindle is engaged in similar functional capacities in the two groups of muscle, leading one to the very obvious question of whether this is, in fact, the case.

2. The importance of studying peripheral mechanisms in motor control, within the context of understanding motor control in general, has been stated by Stein (1974; 216) in the introduction to his review of peripheral control of movement.

> This review is concerned with the constraints imposed on any central programs

by the peripheral sensory and motor systems involved in controlling mammalian muscles.

This summary is based primarily on work done on the spinally-innervated musculature of non-human mammalians (mostly cats).

3. One caveat must be mentioned here. The silent period technique does nct differentiate the presence of muscle spindles from the presence of receptors other than muscle spindles whose response properties are indistinguishable from those of muscle spindles, under the stimulus conditions described. Some results (to be reported below) indicate that periodontal receptors (Goldberg 1971; Sessle et al 1973), and perhaps other, as yet unidentified, receptors in the oral region (Gcldberg 1972a, b, c; Thexton 1973a, b) have response properties similar to those of muscle spindles.

4. Kugelberg (1952) and Hugelin and Bonvallet (1956) have measured the latency tc be approximately 7.5 msec. Goodwill (1968) measured the latency to be 8.4 msec. Goldberg (1971) measured the latency to be 8-12 msec. Munro and Griffin (1971) present the following values: Latency (females) 6.7±1.5 msec.; (males) 6.2±1.9 msec. Duration (females) 9.0±2.3 msec.; (males) 9.0±1.7 msec. Minimum latency 3.5 msec. Fujii and Mitani (1973) found the latency of the H wave (hhich is electrically induced and thus is not actually a jaw-jerk reflex but does involve the same reflex) to be 6.0 msec and the latency of the T wave (which is evoked by

tapping the zygomatic arch with a hammer and thus is not actually a jaw-jerk reflex either) to be 7.0 msec.

5. These, and similar results to be reported here, demonstrate the difficulty in applying the concepts of agonist and antagonist to masticatory muscles.

6. Schaerer et al (1967) measured the latency of the first period of EMG inactivity following tooth tapping during centric occlusion to be 25-60 msec. They found its latency to be more variable during chewing. Brenman et al (1968) measured the latency to be 6-35 msec with a median range of 16-20 msec and the duration to be 5-50 msec and during biting to be 15.3 msec. He did not measure the latency. Griffin and Munro (1969) measured its latency to be 14.5 msec and its duration to be 13 msec. Hannam et al (1969) found the period of EMG inactivity to have a latency of 12 msec and a duration of 10-20 msec. They confirmed these values in a later (1970) study. Munro and Griffin (1970) measured its latency to be 12-13.7 msec and its duration to be 11.4-13.6 msec. The minimum latency was 5.3 msec. Goldberg (1971) measured the latency of the excitatory response in masseter following tooth tap to be 6.5-8.8 msec (mean 7.5 msec) and the duration of the EMG inactivity following to be 20 msec. Electrical stimulation evoked a similar response at a slightly shorter latency (7.0 msec). Hoffman and Tonnies (1948) report the period of EMG inactivity following electrical sitmulation of the tongue to have a duration of 30-50 msec. Bratzlavasky (1972a, c,

1973) measured the latency of the EMG inactivity response to electrical stimulation of various intraoral sites to be 10-15 msec with a duration of 10-20 msec. Yemm (1972a) measured the latency to be 13-19 msec with a duration of 10-19 msec. He found similar results (latency of 13-18 msec) in a later (1972b) study. Matthews and Yemm (1970) report the latency of the masseteric EMG inactivity response to tooth contact in edentulous subjects to be approximately 12 msec and its duration to be 8-20 msec. Bratzlavasky (1972c) reports the latency of EMG suppression following a weak tap to the perioral skin or intraoral mucosa was 10-15 msec, with a duration of 10-20 msec. Yu et al (1973) found that innocuous mechanical stimulation of the upper lip produced masseteric EMG inactivity with a latency of 15-20 msec and a duration of 8-18 msec.

7. Bratzlavasky found the latency of this response to be 30-60 msec (1972a, c, 1973) and the duration to be 10-50 msec (1972a, c). Yemm (1972a, b) discusses, but does not present values for this second period of EMG inactivity. On the basis of photographs presented in his articles we can estimate its latency to be approximately 40-60 msec. Yu et al (1973) found the latency of this response to be 40-50 msec and the duration to be 15-25 msec. They also report that durations as long as 35 msec were common and that occasionally the duration was up to 55 msec, depending on the voluntary effort of the subject.

8. Munro and Griffin (1971) have dissected and measured the

afferent pathway from the masseter muscle of man to the entry of the trigeminal nerve into the pons. They found the pathway to be 66 mm in length. They estimate the distance from the point of entry of CV into the pons to MSV as 15 mm.

[9]. Determination of the length of the lingual nerve was done by tracing a wire probe through a human skull. This estimate is probably low because "the lingual nerve runs a very tortuous course through the tongue musculature." (Porter 1966; 106).

[10]. Ringel (1962) examined the effective duration of lingual surface anesthesia as a part of his dissertation research concerning effects of oral anesthesia on speech. He concluded that for his purposes the effective duration of anesthesia was three minutes. Our observations are similar to his.

[11]. Selective nerve blocks can also be effected by use of nerve pressure. Large diameter nerves are more susceptible than are small diameter nerves to pressure blocks. In the present discussion we are interested only in anesthetic nerve blocks.

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